DISSERTATION ON

CLINICAL STUDY ON POSTERIOR UVEITIS

Submitted in partial fulfillment of requirements of

M.S.OPHTHALMOLOGY

BRANCH – III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY,

CHENNAI

APRIL 2015

CERTIFICATE

This is to certify that this dissertation titled "**A CLINICAL STUDY ON POSTERIOR UVEITIS**" is bonafide record of the research work done by **DR. KASTHURI. B,**Post graduate in the Regional Institute of Ophthalmology & Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by the Tamil Nadu Dr. M.G.R Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic year 2012 – 2015.

PROF. DR.V. REVATHI M.S., D.O.,
CHIEF, UVEA AND RETINA SERVICES,
Regional Institute of Ophthalmology &
Government Ophthalmic Hospital,
Madras Medical College,
Chennai-600 008.

PROF.DR.K.NAMITHA
BHUVANESWARI M.S., D.O.,
DIRECTOR AND SUPERINTENDENT,
Regional Institute of Ophthalmology &
Government Ophthalmic Hospital,
Madras Medical College,
Chennai-600 008

PROF.DR.R.VIMALA.M.D.,
DEAN
Madras Medical College &
Government General Hospital,
Chennai-600 003.

DECLARATION BY THE CANDIDATE

I hereby declare this dissertation entitled **"CLINICAL STUDY ON POSTERIOR UVEITIS"** is a bonafide and genuineresearch work carried out by me under the guidance of Prof.**Dr.V REVATHI, M.S D.O.,**

DATE : **DR.KASTHURI.B.**

PLACE:

ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to **Prof.Dr.R.Vimala, M.D., Dean,** Madras Medical College, for permitting me to conduct this study.

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I wish to express my sincere thanks to all the professors, assistant professors and all my colleagues who had helped me in bringing out this study.

Finally, I am indebted to all the patients for their sincere co-operation for the completion of this study.

INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
Dr. Kasthuri .B,
Post Graduate,
Regional Institute of Ophthalmology and Govt. Ophthalmic Hospital,
Madras Medical College, Chennai – 600003.

Dear Dr. Kasthuri .B,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Clinical study on posterior uveitis"** No.44062014

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
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11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

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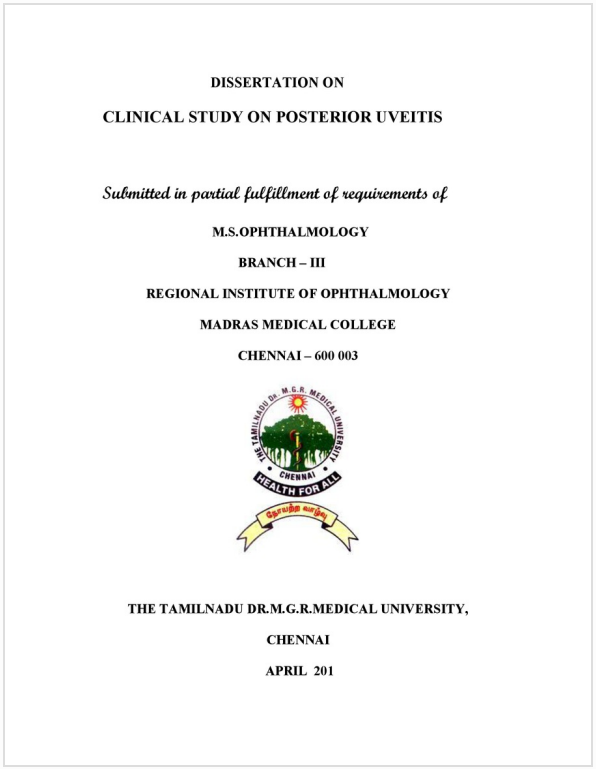

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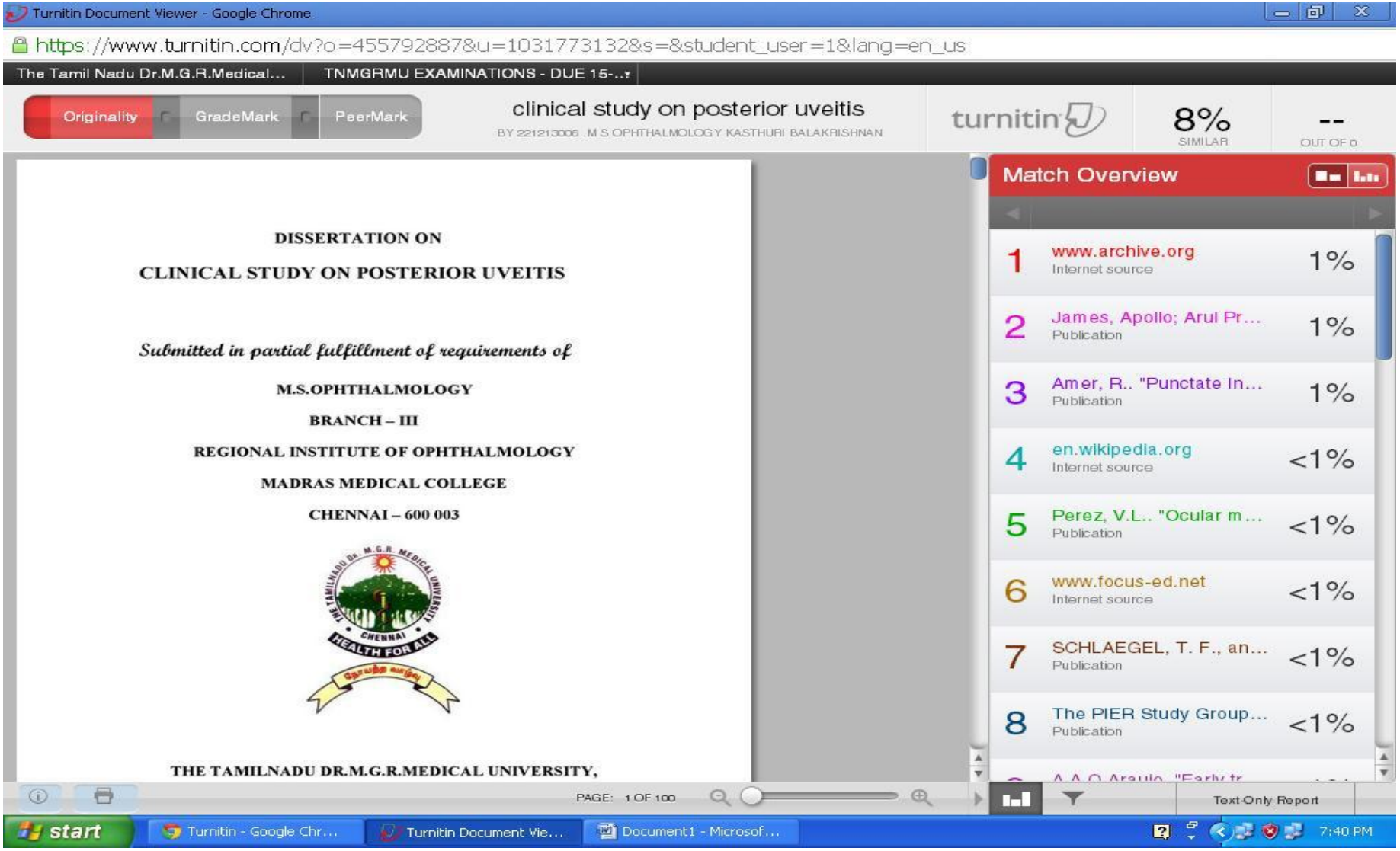
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PART I

CLINICAL STUDY ON POSTERIOR UVEITIS

INTRODUCTION

Posterior Uveitis refers to inflammation of retina and or choroid. Uvea the middle vascular coat of eye consists of iris, ciliary body and choroid.

Posterior uveitis is less common than anterior uveitis.The approximate incidence of 10 - 20 % can be extrapolated from the literature.

Posterior uveitis with its varied aetiology, varied clinical picture, associated multiple sequelae and complications signifies the early diagnosis and prompt treatment. However the disease is associated with frequent relapses and more protracted course often presenting a clinical challenge to the treating Opthalmologist,as it is known for gradual visual loss.

ANATOMY OF THE UVEAL TRACT

Uvea is the middle vascular coat of the eyeball, consists of iris, ciliary body and choroid. The term uvea derived from latin word uva (grape).

Iris

Iris is the anterior most part of the uveal tract and forms the pupillary diaphragm of the eye. The anterior surface of iris is divided as small pupillary zone and large peripheral ciliary zone by the collarette or minor arterial circle, the thickest part of the iris[7]. The thinnest most peripheral portion of the iris, iris root inserted to the middle of the anterior surface of the ciliary body.

The microscopic structure of iris consists of anterior limiting layer, iris stroma, anterior pigmented epithelial layer and posterior non pigmented epithelial layer. Iris stroma consists of two smooth muscles sphincter pupillae in the pupillary zone and dilator pupillae in the ciliary zone.

Ciliary body

The ciliary body is the middle part of the uveal tract between iris and choroid. Pars plicata (Corona ciliaris) containing finger like ciliary processes is the anterior one third of ciliary body. Pars plana (Orbicularis ciliaris) is the smooth posterior two thirds of ciliary body.

The microscopic structure consists of supra ciliary lamina, ciliary body stroma, pigmented epithelial layer, non pigmented epithelial layer and internal limiting membrane. Ciliary muscle is a non-striated muscle consists of longitudinal,circular and radial fibres.

The major arterial circle located in the stroma of the ciliary body and supplies iris, ciliary body and anterior choroid.

Choroid

The choroid is the posterior most part of the uvea that extends from optic discto ora serrata between RPE and sclera. The microscopic structure consists of suprachoroidal lamina,stroma, choriocapillaries and bruch's membrane .

The stroma consists of collagen, elastic and reticular fibres, melanocytes, macrophages,mast cells, lymphocytes, plasma cells and

vessels arranged in two layers as outer layer of large vessels(Haller's layer), inner layer of medium vessels(Sattler's layer) that supply and drain choroid.

Choriocapillaries consists of a rich capillary network which receives blood from large and medium vessels of stroma. Capillaries contain fenestrated endothelial cells that allow passage of nutrients to RPE and outer layers of sensory retina.

Bruch's membrane (lamina vitrae) is the innermost layer of choroid. It is a multilayered structure that lies between the choriocapillaries and retinal pigment epithelium.

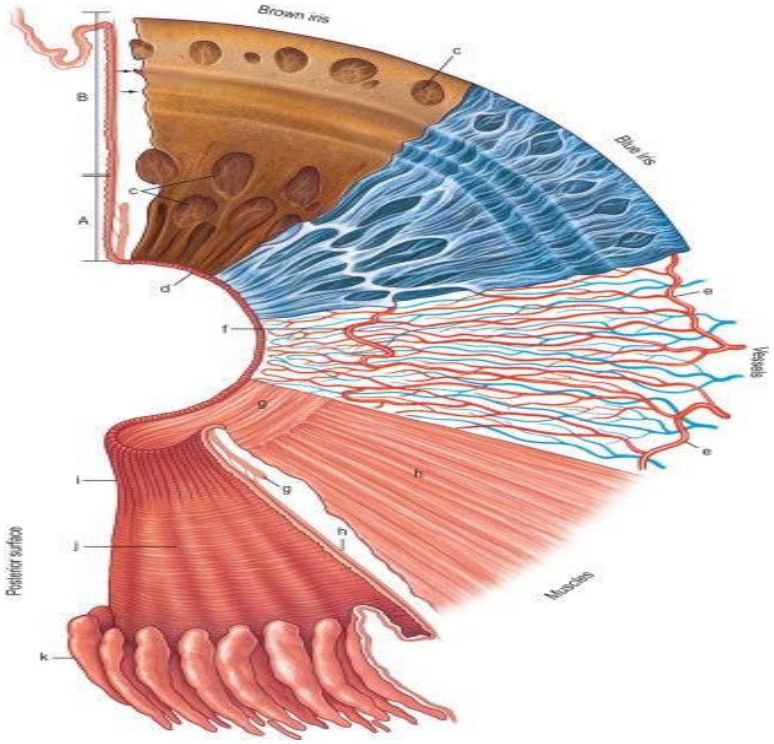
Vitreous Humour

It is a transparent gel that provides a clear optical medium, structural integrity to the eye and a pathway for nutrients utilized by the lens, ciliary body and retina. Vitreous is clear and avascular, filling the space bound by the lens, retina and optic disc. It occupies approximately 80% of the globe volume. The vireous consists of largely of water (99%), a network of collagen fibrils, hyaluronic acid, hyalocytes and mucopolysaccharides, forming a gel like material.

Retina

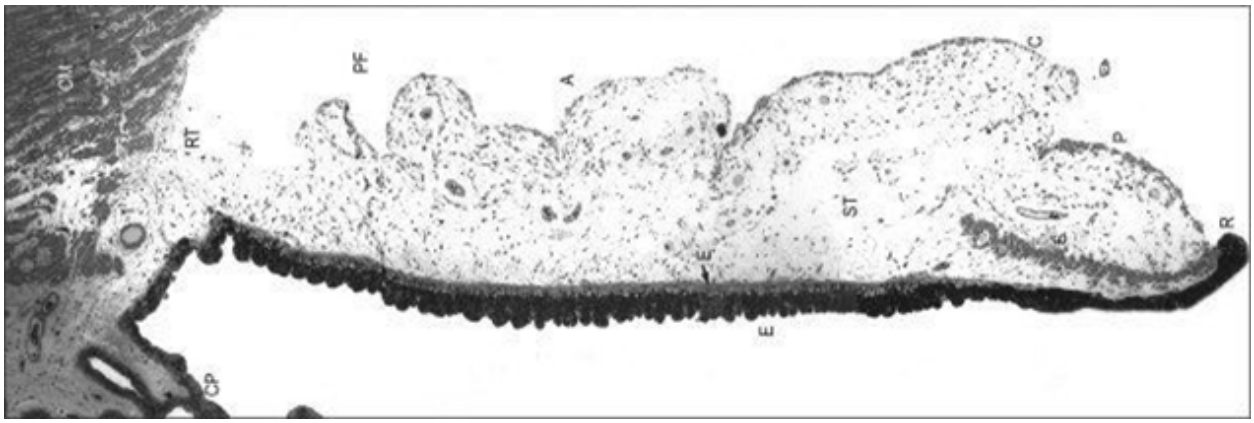
It is the innermost layer of eyeball, a thin delicate and transparent membrane. Retina extends from the optic disc to ora serrata. Grossly on ophthalmoscopic examination it can be divided into three distinct regions namely optic disc, macula lutea and peripheral retina. Microscopic structure of retina consists of 3 types of cells (rods and cones, bipolar cells, ganglion cells) and their synapses arranged in 10 layers.

Outer four layers of retina viz. retinal pigment epithelium, layers of rods and cones, external limiting membrane and outer nuclear layer get their nutrition from the choriocapillaries. Inner six layers get their supply from the central retinal artery. The fovea is an avascular area mainly supplied by the choriocapillaries. Inflammation of choroid always involves the retina secondarily.



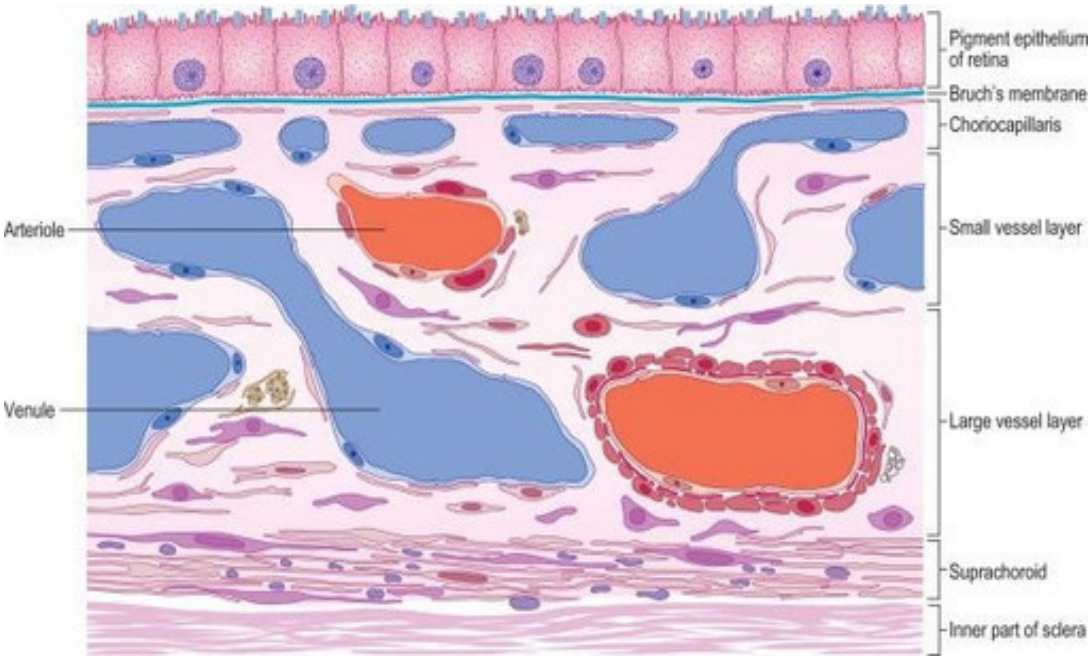
STRUCTURE OF IRIS

HISTOLOGY OF IRIS TRANSVERSE SECTION



STRUCTURE OF CHOROID AND RETINA

HISTOLOGY OF CHOROID



HISTORICAL REVIEW

1500 BC - Von Hippocrates mentioned the typical findings of uveitis.

12th century - Mohammed-al-Ghafiqi described a disease with poliosis, neuralgia and hearing changes.

19th century - Mackenzie described about poor vision after couching for cataract in same eye and fellow eye

1650- 1730 - Antoine Maitre Jan described choroiditis for the first time.

1881 - Von Michael emphasizedthe importance of Tuberculosis in uveitis.

1900 - Neetleship considered a case of exudative choroiditis to be due to dental infection.

1906 - Alfred Vogt and 1929 Koyanagi described bilateral nontraumatic chronic iridocyclitis associated with poliosis, vitiligo and dysacousia.

1926 - Harada described posterior uveitis with exudative retinal detachment associated with CSF pleocytosis.

1924 - Shigeta in Japan, 1931- Adamantiades in French and 1937 – Hulsi Bechet reported a multisystem disorder

with triad of symptoms (recurrent ocular inflammatory episodes, oral and genital leisons)

1936 - Sarcoid uveitis associated with facial nerve palsy and uveoparotid fever was termed as Heerfordt's syndrome.

1940 - Brucellosis and Sarcoidosis were recognized as clinical entities.

1946 - Allan C Wood considered that 75% of granulomatous uveitis was due to Tuberculosis.

1950 - Toxoplasma uveitis became a proven infection, and thus parasitic infection played a considerable role in the aetiology particularly of posterior uveitis.

1950 - Wilder reported nematode larva in eyes enucleated for retinoblastoma and Nichols determined the cause is toxocara canis.

1952 - Helenor Campbell Wider identified the Toxoplasma Gondii in eyes and confirmed it was the cause for uveitis

1961 - Wood considered Histoplasmosis to be the cause of 13% of uveitic cases.

EPIDEMIOLOGY

The incidence of posterior uveitis is less common than anterior uveitis. Infectious causes are more common in posterior uveitis. Toxoplasmosis is the most common cause of infectious retinitis in immunocompetent individuals. CMV retinitis is the most common opportunistic ocular infection among AIDS patients.

Age incidence

Posterior uveitis can occur in all age groups. The etiology differs in each age group. Toxocariasis, congenital Toxoplasmosis are common in children. Toxoplasmosis, white dot syndromes and VKH syndrome are common in 20-60years. Idiopathic retinal vasculitis and masquerade syndromes occurin more than 60 years of age[2].

Sex incidence

Males are more commonly affected in sympathetic ophthalmia, Eales disease. Females are more affected in Rheumatoid Arthritis, Systemic Lupus Erythematosus associated uveitis. There is no gender predilection for infectious uveitis.

Duration and laterality

Majority of posterior uveitis is of chronic duration. Most cases of posterior uveitis are bilateral. Parasitic diseases are typically unilateral.

PATHOGENESIS OF UVEITIS

a) Direct Bacterial invasion

One of the oldest and major hypothesis is the concept of direct bacterial invasion. Another hypothesis for recurrent ocular inflammation assumes a structural alteration within the eye resulting from a previous inflammation that predisposes the to recurrent inflammatory episodes[2].

b) Prostaglandins

Prostaglandin is a chemical mediator involved in the pathogenesis of uveitis. Prostaglandin being synthesized by the enzyme prostaglandin synthetase, cause a dramatic increase in protein content and flare of aqueous humor and mild smooth muscle contraction (miosis).

c) The uvea as a node

When the antigen is introduced at distant site or is injected directly into the circulation, antibody production begins inside the eye, which takes up residence within the uvea and remain for long period. Here the new antigen is needed to stimulate them for the renewal of the antibody response.

d) Vitreous Antigen depot

Once antigen gain access to vitreous body,it tends to persist and prolong immune responseto trapped antigen. Since hyalocytes have macrophagic character, they may promote a persistent tendency to recurrence of uveitis by processing antigen and modulating the immune responses.

e) Focal infection

Any focal sepsis is predisposing factor for uveitis since most of ocular inflammation are infective in nature. Among the foci of infection the teeth is considered the most profile source. The others being the tonsils, paranasal sinuses, respiratory tract, alimentary tract, uterus and urinary tract.

f) Immune response

The major focus now is on the immune characteristics of the eye. Absence of lymphatic drainage in the eye has special features and plays a role in the Anterior Chamber Associated Immune Deviation (ACAID). Uveitis wherein hypersensitivity is dominant, 3 types of reactions must be considered.

1) **Immediate Hypersensitivity (Anaphylaxis**)

This results from contact of the uveal tissue to some foreign protein namely bacterial protein, where an immediate antigen antibody reaction occurs.

2) Delayed Hypersensitivity (Bacterial Allergy)

The tissues are sensitized by contact with living or dead organism so that antibodies are formed on or within cells. Further contact with same antigen causes severe cellular damage which develops slowly causing an inflammatory and necrotic tissue reaction causing allergic iridocyclitis.

3) Immune complex mediated disease

Immune complexes can be demonstrated in the aqueous of patients with uveitis.e.g, Bechet's disease. These findings have led to the speculation that immune complex mediated tissue destruction could explain intraocular inflammatory disease. Serum complement levels have also been low in such patients.

g) Autoimmunity

It is an immune response directed against the host. Example is autoantibody production to the lens. The presence of uveitogenic antigens in the eye that are capable of inducing disease is an old concept proposed as early as 1910 by Elschnig. The retina contains these antigens (SAg) and it is also particularly prone to certain neurotropic organisms like Toxoplasma Gondii and Herpes viruses.

h) Histo compatibility antigen of uveitis

HLA system is now regarded as the main leucocyte in antigen system. Some special types of anterior uveitis has been associated with inflammatory bowel disease like Ulcerative colitis, Crohn's disaease, the useful indicator being HLAB27. So there is considerable genetic variability in susceptibility to the diseases.

CLASSIFICATION OF UVEITIS

Anatomical classification (IUSG)

Anterior uveitis	-	Iritis
	-	Iridocyclitis
	-	Anterior cyclitis
Intermediate uveitis	-	Posterior cyclitis
	-	Pars planitis
	-	Hyalitis
	-	Basal retinochoroiditis
Posterior uveitis	-	Choroiditis (focal/multifocal/diffuse)
	-	Chorioretinitis
	-	Retinochoroiditis
	-	Retinitis
	-	Neuroretinitis
	-	Retinal vasculitis
Panuveitis		

Clinical classification

Laterality	-	Unilateral
	-	Bilateral
Severity	-	Mild
	-	Moderate
	-	Severe
Chronicity	-	Acute
	-	Acute recurrent
	-	Chronic
Pattern	-	Focal
	-	Multifocal
	-	Diffuse
Pathology	-	Granulomatous
	-	Non granulomatosus
Demographics	-	Age
	-	Sex
	-	Race

Aetiological classification

a) Exogenous uveitis : By external injury to the uvea or invasion of microorganisms or other agents from outside.

b) Endogenous uveitis : By microorganisms or other agents from within the patient.

POSTERIOR UVEITIS

Posterior uveitis is defined as intraocular inflammation primarily involving retina and or choroid. In posterior uveitis inflammatory cells may be observed diffusely through out vitreous cavity, overlying foci of activeinflammation or in the posterior vitreous face. Macular edema, retinal vasculitis and retinal or choroidal neovascularization are the structural complications of posterior uveitis[6].

Posterior uveitis encompasses

a) Retinitis

b) Choroiditis

c) Retinal vasculitis.

Retinitis

It may be focal, multifocal, geographic or diffuse. Active leisons characterized by whitish retinal opacities with indistinct border due to surrounding edema with overlying vitreous cells. As the lesion resolves the borders become better defined.

Choroiditis

It may be focal, multifocal, geographic or diffuse. Active choroiditis is characterized by a round yellow nodule. It does not usually induce vitritis in the absence of concomitant retinal involvement. Old inactive disease appear as atropic chorioretinal leisons with surrounding hyperpigmentation.

Retinal Vasculitis

It may occur as a primary condition or as a secondary phenomenon adjacent to a focus of retinitis. Veins are more commonly involved than arteries. Active vasculitis is characterized by yellow or grey white, patchy perivascular cuffing, retinal hemorrhages and cotton wool spots. Quiescent vasculitis have perivascular scarring, atropic retina and pigment epithelial stippling.

Aetiology of posterior uveitis

1) Non infectious causes

Collagen vascular diseases – Systemic Lupus Erythematosus, Polyarteritis nodosa, Microscopic polyangitis and Wegener granulomatosis.

2) Inflammatory chorioretinopathies of unknown etiology

Inflammatory chorioretinopathies or White dot syndromes are a heterogenous group of inflammatory disorders with overlapping clinical features. These include

a) Birdshot retinochoroidopathy

b) Acute posterior multifocal placoid pigment epitheliopathy

c) Serpiginous choroiditis

d) Multifocal choroiditis and panuveitis

e) Punctate inner choroiditis

f) Subretinal fibrosis and uveitis syndrome

g) Multiple evanescent white dot syndrome

h) Acute retinal pigment epithelitis

i) Acute zonal occult outer retinopathy

3) Infectious causes

a) **Viral infections** - Herpes simplex and Varicella zoster virus, Cytomegalovirus, Epstein Barr virus, Rubella, Measles, Lymphocytic choriomeningitis virus, Westnile virus, Rift valley fever, Human T cell lymphotrophic virus, Dengue fever and chikungunya fever

b) **Bacterial infections** – Tuberculosis, syphilis, Lyme disease, Leptospirosis, Bartonellosis and Whipple disease

c) **Fungal infections** – Ocular histoplasmosis syndrome, Candida, aspergillus and Cryptococcus

d) **Protozoal infection** – Toxoplasmosis

e) **Helminthic infections** – Toxocariasis, cysticercosis, Onchocerciasis and Diffuse unilateral subacute neuroretinitis

4) Post traumatic uveitis

5) Post surgical uveitis

Symptoms of posterior uveitis

Floaters and defective vision are the most common symptoms of posterior uveitis. Floaters result from the shadows cast by vitreous cells and opacities on the retina.

Defective vision may be caused by the primary effects of uveitis, such as retinitis or choroiditis directly affecting the macular function or by the complications of inflammation such as cystoid macular edema, epiretinal membrane,retinal ischemia,choroidal neovascularization, opacities in the visual axis from inflammatory cells,fibrin, secondary cataract, myopic or hyperopic shift from macular edema, protein in anterior chamber and keratic precipitates inpanuveitis or spill over anterior uveitis.

Symptoms due to anterior uveitis such as pain, redness, photophobia, watering and decreased vision.

Signs of uveitis

In Anterior segment

Keratic precipitates

Inflammatorycells

Flare

Hypopyon

Pigment dispersion

Pupillary miosis

Iris nodules

Synechiae (anterior and posterior)

Band keratopathy (long standing cases)

SUN working group grading of Anterior Chamber cells

Cells in field	Grade
<1	0
1-5	+-
6-15	1+
16-25	2+
26-50	3+
>50	4+

Grading of aqueous flare

Description	Grade
Nil	0
Just detectable	1+
Moderate (iris & lens details clear)	2+
Marked (iris & lens details hazy)	3+
Intense (fibrinous exudate)	4+

Keratic precipitates (KPs)

These are small aggregates of inflammatory cells that accumulate on the endothelial surface on cornea. It indicates current level of inflammatory activity, mostly found in lower half of cornea in a base down triangle configuration between 4 and 8'o clock position an area termed as Arlt's triangle[2].

KPs are categorized on the basis of

Size - small / medium / large

Shape and colour - white and round (fresh)

- Shrunken and pigmented (old)

Appearance - mutton fat KPs (Granulomatous)

- White and round (Non granulomatous)

Perfield	**Grading**
5-10	1+
11-20	2+
20-50	3+
>50	4+

Signs in posterior segment

Retinal or choroidal inflammatory infiltrate

Inflammatory sheathing of arteries or veins

Exudative, tractional or rhegmatogenous retinal detachment

Retinal pigment epithelial hypertrophy or atrophy

Swelling or atrophy of retina, choroid or optic nerve head

Preretinal or subretinal fibrosis

Retinal or choroidal neovascularization

Grading of vitreous cells (Hruby lens)

Cells in retroilluminated field	**Description**	**Grade**
0-1	Clear	0+
2-20	Few opacities	Trace
21-50	Scattered opacities	1+
51-100	Moderate opacities	2+
101-250	Many opacities	3+
>251	Dense opacities	4+

Examination of the anterior vitreous is done with a slit lamp by rotating the slit 45 degrees and rotating the slit or illumination arm at the minimum angle of separation from the viewing pathway.

Vitreous haze is a better indicator of active inflammation than are vitreous cells because it combines the optical effect of cellular infiltration and protein leakage. Grading of vitreous haze is done by comparing the patient examination by indirect ophthalmoscope with standard colour photograph in the examination room.

Grading of vitreous haze

Haze severity	Grading
Good view of nerve fibre layer	0
Clear disc & vessels but hazy NFL	1+
Disc and vessels hazy	2+
Only disc visible	3+
Disc not visible	4+

Examination of Retina and Choroid

Examination of retina and choroid are done with combination of indirect ophthalmoscope, Hruby lens, + 90 diopter lens, mirrored contact lens. The indirect ophthalmoscope is ideal for defining the extent and height of retinal and choroidal leisons, to find out peripheral vascular sheathing, narrowing, obliteration and deep isolated choroidal leisons. Hruby lens +90 diopter lens are useful to determine the depth of the lesion. Mirrored contact lens are useful to look for the midperiphery of retina.

Complications of posterior uveitis

Cystoid Macular edema (CME)

It is a common cause of visual loss in posterior uveitis . Macular edema is caused by active intraocular inflammation that leading to retinal vascular leakage and retinal pigment epithelial dysfunction. CME is quantitatively evaluated and followed by serial spectral domain OCT and Fundus Fluorescein Angiography.

A recent study of uveitic macular edema divided the edema morphologically into diffuse macular edema, cystoid macular edema and serous retinal detachment.

Retinal detachment

Rhegmetogenous retinal detachment and Tractional retinal detachment are common in posterior uveitis. Infectious uveitis and panuveitis are more frequently associated with rhegmatogenous retinal detachment(RRD).Uveitis is usually still active with retinal detachment. Upto 30% of patients with RRD may have proliferative vitreoretinopathy at presentation. Repair is difficult due to preexisting PVR, vitreous organization and poor visualization.

Vitreous opacification and Vitritis

Permanent vitreous opacification affects the vision. It is treated with 3- port pars plana vitrectomy.

Proliferative retinopathy

Retinal neovascularization occurs from chronic inflammation or capillary non perfusion. It is commonly seen in retinal vasculitis of various causes and sarcoid panuveitis.

Choroidal neovascularization

Choroidal neovascularization results from a disruption of the Bruch membrane from choroidal inflammation and the presence of inflammatory cytokines that promote angiogenesis. It is commonly occur in ocular Histoplassosis syndrome, puntate inner choroidopathy, idiopathic multifocal choroiditis and serpiginous choroiditis. It can be of foveal, juxta foveal, extrafoveal and peripapillary types.

Other complications due to spill over anterior uveitis

Secondary glaucoma

Complicated cataract

Hypotony

Aetiologyof posterior uveitis

Ocular Toxoplasmosis

Toxoplasmosis is the most common cause of infectious retinochoroiditis in immunocompetent adults and children. Classically presents as new unilateral white yellow retinal lesion with overlying moderate vitreous inflammation (headlight in the fog), often adjacent to a pigmented chorioretinal scar with perivasculitis and diffuse venous sheathing of adjacent retinal vessels. Most of the leisons occurs in the posterior pole.

Diagnosis is made clinically and serological evaluation using ELISA test used to confirm the exposure to parasite. Treatment regimen is classical triple therapy (systemic prednisolone, pyrimethamine and sulfadiazine). Alternative regimens are co-trimaxozole or azithromycin with systemic corticosteroids.

Tuberculosis

Uveitis is the most common manifestation of secondary ocular TB. Tuberculous uveitis is classically a chronic granulomatous disease that affect anterior and posterior segments.

Disseminated choroiditis is the most common presentation characterized by deep, multiple, discrete, yellow leisons between 0.5 and 3 mm diameter located predominantly in posterior pole associated with disc edema, vitritis, granulomatous anterior uveitis. Other presentation single, focal, large, elevatedchoroidal mass (tuberculoma) of 4-14 mm in size.

Other posterior segment findings of TB includes subretinal abscess, CNV, optic neuritis and acute endophthalmitis. Retinal involvement present as Eales disease and peripheral retinal perivasculitis.

Cytomegalovirus retinitis

Cytomegalovirus is an opportunistic pathogen in immunocompromised individuals and the most common infection in AIDS typically occurs when the CD4 count is <50 cells/microliter. Cytomegalovirus retinitis is characterized by greyish patches or scaterred white dots with irregular sheathing of adjacent blood vessels. There are superimposed haemorrhages followed by healing and retinal atrophy. Ganciclovir is used in the treatment of CMV retinitis.

Toxocariasis

Ocular toxocariasis is an uncommon disease of children and young adults that causes significant visual loss. It is acquired by ingestion of soil containing eggs of the canine intestinal round worm Toxocara canis. It otherwise involves healthy individualswith a normal white cell count and absence of eosinophilia. It is unilateral and in children it presents as chronic endophthalmitis and posterior pole granuloma. In adolescence or adult life it presents as peripheral retinal granuloma associated with a vitreous band extending to the disc or macula causing visual impairment.

Lab diagnosis is by the high sensitivity and specificity (90%) of an ELISA titre for Toxocara. Oral or periocular corticosteroids are used for treatment.

Noninfectious autoimmune diseases

These are the collagen vascular diseases namely Systemic lupus erythematotosus , Polyarteritis nodosa and Microscopic polyangitis and Wegener granulomatosis.

Systemic lupus erythematosus

It is a connective tissue disorder with multisystem involvement that primarily affects women of child bearing age. Ocular involvement occur in 50% of cases and include cutaneous leisons on eyelids, sjogren syndrome, scleritis, cranial nerve palsies and retinal vasculopathy.

Lupus retinopathy is an important marker of systemic disease activity. The clinical spectrum is characterized by cotton wool spots with or without intraretinal hemorrhages due to microangiopathy, severe retinal vascular occlusive disease resulting in retinal nonperfusion and ischemia, secondary retinal neovascularization and vitreous hemorrhage, lupus choroidopathy resulting in serous elevations of retina, choroidal infarction and choroidal neovascularization.

The diagnosis is essentially clinical based on revised diagnostic criteria. Antinuclear antibody anti- ds DNA antibody (anti double stranded deoxyribo nucleic acid antibody) and antiphospholipid antibodies are positive in SLE.

Treatment is control of underlying disease with NSAIDS (Non steroidal anti-inflammatory drugs), Corticosteroids and

immunosuppressive drugs. Ischemic complications are managed with panretinal photocoagulation and vitrectomy surgery.

Polyarteritis nodosa and microscopic polyangitis

Polyartertis nodosa is an uncommon systemic vasculitis characterized by subacute or chronic, focal,episodic necrotizing inflammation of medium sized and small muscular arteries. It is seen in patients between the ages of 40-60 years. Ocular involvement seen in 20% of patients includes hypertensive retinopathy, retinal arteriolar occlusive disease, choroidal infarcts with exudative retinal detachment secondary to vasculitis involving posterior ciliary arteries and choroidal vessels, cranial nerve palsies and peripheral ulcerative keratitis with scleral involvement. pANCA (perinuclear antineutrophil cytoplasmic antibody)is positive in Polyarteritis nodosa and microscopic polyangitis.Treatment consists of systemic corticosteroids and immunomodulators[6].

Wegener granulomatosis

It is a multisystem autoimmune disorder characterized by the classic triad of necrotizing granulomatous vasculitis of upper and lower respiratory tract, focal segmental glomerulonephritis and necrotizing

vasculitis of small arteries and veins. Ocular or orbital involvement seen in 15% of cases. Orbital involvement is secondary to contiguous extension of the granulomatous inflammatory process from the paranasal sinuses.

Ocular involvement includes unilateral or bilateral anterior, intermediate or posterior uveitis, with varying degrees of vitritis and retinal vasculitis leading to retinal neovascularization, vitreous hemorrhage and neovascular glaucoma. c ANCA (cytoplasmic antineutrophil cytoplasmic antibody)positivity is sensitive and specific for Wegener granulomatosis. Treatment includes systemic corticosteroids and immunosupressants.

Eales disease

It is an idiopathic, inflammatory peripheral retinal vasculopathy which presents with recurrent vitreous hemorrhages in young males. It has been suggested that a hypersensitivity of retinal vessels to tuberculoproteins may be the cause for retinal vasculitis. The vasculitis leads to obliteration of affected vessels, subsequent hypoxia and vasoproliferation.

It is clinically seen as sheathing of the vessels, which leaks copiously on FFA. Hemorrhages , soft exudates and retinal edema are common at the junction of perfused and non perfused zones of retina. In proliferative stage it presents as recurrent vitreous hemorrhages, vitreous traction and retinal detachment.

Treatment of acute vasculitic stage consists of systemic steroids. Proliferative stage is treated with retinal photocoagulation and vitreoretinal surgery.

White dot syndromes

The inflammatory chorioretinopathies or white dot syndromes are a heterogenous group of inflammatory disorders with overlapping clinical features that share in common the presence of discrete, multiple, well circumscribed, yellow white leisons at the level of retina, outer retina, RPE, choriocapillaries and choroid during some phase of their course. These consists of predominantly non infectious ocular syndromes.

Common presenting symptoms include photopsias, blurred vision, nyctalopia, floaters and visual field loss contiguous with a blind spot. The syndromes are bilateral and asymmetrical in nature. The etiology of

the white dot syndromes is unknown. Infectious, autoimmune and infiammatory pathogenesis has been postulated for these disorders.

Panuveitis

Panuveitis is defined as inflammation involving all anatomical compartment of eye (anterior chamber, vitreous and retina or choroid) with no single predominant site of inflammation.It is usually bilateral. Common causes are

a) Vogt – Koyanagi – Harada syndrome

b) Sarcoidosis

c) Sympathetic ophthalmia

d) Behcet disease

e) Infectious causes – Tuberculosis, Syphilis

Vogt –Koyanagi-Harada syndrome

It is an uncommon multisystem disease of presumed autoimmune etiology that is characterized by chronic, bilateral, diffuse granulomatous panuveitis with accompanying integumentary,

neurological and auditoryinvolvement. There are four stages of VKH syndrome: prodromal, acute uveitic, convalescent and chronic recurrent.

Acute uveitic stage characterized by sequential blurring of vision in both eyes with bilateral granulomatous anterior uveitis,variable degree of vitritis, thickening of the posterior choroid, hyperemia and edema of disc and multiple serous detachments. The chronic phase shows diffuse RPE atrophy (sunset glow fundus).Complications include CNV and subretinal fibrosis.

FFA of the acute phase shows multifocal hyperfluorescent dots at the level of RPE and then accumulation of dye in the subretinal space. The chronic phase shows are areas of hyperfluorescence due to RPE window defects.

Treatment involves high dose oral prednisolone (60-100mg/day) that may be augmented with 3 days intravenous pulse therapy with methyl prednisolone (500-1000mg/day).Steroid resistant patients require immunosupreesive therapy. Topical Corticosteroids and topical cycloplegics for anterior uveitis.

INVESTIGATIONS

There is no one standardized battery of tests that need to be ordered for all patients with posterior uveitis. Rather a tailored approach should be taken based on the most likely causes for each patient. Once a list of differential diagnosis is compiled appropriate laboratory tests can be ordered. Many patients require only one or a few diagnostic tests[6].

If the history and clinical examination do not clearly indicate any cause, purified protein derivative skin test, chest radiograph, chest computed tomography, serum angiotensin converting enzyme and syphilis serologies are done to rule out the most common causes (Tuberculosis, Syphilis and Sarcoidosis).

Laboratory tests and imaging studies with indications

Test	Indications
HEMATOLOGICAL TESTS	
Complete blood count	Immunomodulator therapy
Erythrocyte sedimentation rate	Gaint cell arteritis
Quatiferon gold	Tuberculosis
T cell subsets	AIDS
SEROLOGICAL TESTS	Indications

Liver function test	Immunomodulator therapy, sarcoidosis
Renal function test	Immunomodulator therapy
Angiotensin converting enzyme	Sarcoidosis
Antiphospholipid antibodies	Vascular occlusion
Rheumatoid Factor	Rheumatoid arthritis, juvenile idiopathic arthritis
Antinuclear antibody	Connective tissue diseases
HLA testing	
HLA-B27	Seronegative spondyloarthropathy
HLA-A29	Birdshot retinochoroidopathy
HLA-B51	Bechet disease
ANCA (c ANCA and p ANCA)	Systemic vasculitides
VDRL/RPR	Syphilis
FTA ABS	Syphilis
Toxoplasma antibodies	Toxoplasmosis
HSV,VZV,CMV serology	Viral uveitis
HIV serology/ Westernblot	HIV/AIDS
Radiographic studies	Indications
Chest radiograph	Tuberculosis, sarcoidosis, wegener granulomatosis
Sacroiliac joint	Ankylosing spondylitis
CT chest	Sarcoidosis
CT/MRI brain and orbits	Sarcoidosis, toxoplasmosis

Complete blood count and differential count

It is done to determine whether there isacute or chronic inflammation. Complete blood count is obtained before initiating systemic therapy with either corticosteroid or immunosuppressive drugs.

Erythrocyte sedimentation rate

It is a non-specific indicator of plasma fibrinogen and globulin levels and may be elevated in systemic infection or inflammation or malignancy. It is done using Westergren method.

Rheumatoid Factor

Rheumatoid Factor (RF) is an autoantibody directed against the Fc fragment of human IgG. About 80% of Rheumatoid Arthritis patients are RF seropositive, defined as a titre of more than 1:80. RF seropositivity is non specific and best to support a clinical diagnosis of Rheumatoid Arthritis.

Antinuclear Antibodies (ANA)

The ANA test is typically performed by applying serial dilutions of the patients serum to cultured tumor cells and then titrating for the presence and pattern of nuclear antibody staining. The ANA test is

generally used to confirm collagen vascular disease particularly Systemic Lupus Erythematosus or Juvenile Rheumatoid Arthritis.

Herpes Virus antibodies

The prevalence of Herpes virus antibodies is so high in the general population that a positive antibody titre is virtually meaningless. A negative titre , however eliminates herpes infection and therefore can be useful in selected instances.Herpes virus serology should remain positive for life.

HIV antibodies

Most commonly detected by using an Enzyme Linked Immuno Sorbent Assay (ELISA), positive results are confirmed by a westernblot test. HIV testing in uveitis is usually orderedin patients with known HIV risk factors, severe or bilateral retinitis or choroiditis. HIV testing requires patient consent.

Toxoplasma antibodies

Tests available to detect and quantify antitoxoplasma gondi antibodies are sabin Feldman dye test(SF), Immuno Fluorescence Antibody (IFA) test and ELISA. Of these SF dye test remains the most

sensitive and specificbut it is technically difficult and of limited availability, where as IFA ana ELISA are relatively easy and economical and can be used to distinguish IgG and IgM anti Toxoplasma gondii antibodies. When interpreting positive titres, it is important to remember that IgM anti Toxoplasma antibodies may be elevated for upto 1 year after infection, limiting the accuracy with which they can date acute infection and that antibody titres are generally less reliable in patients with AIDS.

Angiotensin Converting Enzyme level (ACE)

ACE is produced primarily by capillary endothelial cells, abundant in both lungs and liver, and by macrophages. Clinically, ACE levels are elevated in more than two thirds of patients with a active disease of sarcoidosis.

Uveitis and negative Purified Protein Derivative (PPD) with increased ACE is fairly specific for sarcoidosis. Normal serum ACE level is 12-55mol/min/ml in men and 11-19 mol/min/ml in women.

Human Leucocyte Antigen (HLA) study

The surface membrane of human leucocyte contain HLA. These are regulated by gene loci on chromosome 6. The reaction of these

antigens with specific antisera cause lysis of the cell membrane and this is the basis of cell typing. HLAB27 has been associated with Ankylosing spondylitis and Reiter's syndrome.

Mantoux test

Purified protein derivative of tuberculin is injected intradermally. It is a non-specific test. Because of prior exposure to tuberculosis, a large number(8 to 30%) of healthy adults have positive PPD skin test representing inactive infection. Therefore it is disadvantageous as it yields more false positive than true positive results.

X ray chest and X ray sacroiliac joints

Chest x ray is taken in patients suspected by having sarcoidosis or tuberculosis. X ray sacroiliac joints is taken in patients also complaints of joint pains and hip pains.

Fundus Fluorescein angiography

It is an essential modality for evaluation of posterior uveitis. It is useful for both diagnosis and monitoring patient's response to therapy. Areas of choroidal, retinal and optic nerve inflammation, cystoid

macular edema, retinal vasculitis, secondary choroidal and retinal neovascularization are detected angiographically.

Ultrasonography

It is useful in demonstrating vitreous opacities, choroidal thickening, retinal detachment or cyclitic membrane formation, particularly if media opacities preclude a view of posterior segment[6].

Optical coherence tomography

It is used for the objective measurement of uveitic cystoid macular edema, retinal thickening, subretinal fluid associated with choroidal neovascularization and serous retinal detachment.

Indocyanine green angiography

It shows 2 patterns of hypofluorescence in inflammatory choroidal vasculopathies. Type 1 in inflammatorychoriocapillaropathies demonstrate early and late multifocal areas of hypofluorescence. Type 2 represents stromal inflammatory vasculopathies of choroid and demonstrates areas of early hypofluorescence and late hyperfluorescence.

TREATMENT

Mydriatic and Cycloplegic agents

Topical mydriatic and cycloplegic agents are beneficial for breaking or preventing theformation of posterior synechiae and for relieving photophobia secondary to ciliary spasm in spill over anterior uveitis or panuveitis. The stronger the inflammation, the stronger or more frequent the dose of cycloplegic. Short acting drugs such as cyclopentalate hydrochloride 1% or long acting drops such as atropine 0.1% may be used.

Corticosteroids

Corticosteroids are the mainstay of posterior uveitis therapy. It may given in topical, periocular or systemic routes.

Topical steroids

Topical steroids are effective primarily for anterior uveitis. 1% prednisolone acetate eye drops started as hourly, once the inflammation is controlled , frequency gradually tapered to 2 hourly, 3 hourly then 4 times a day, eventually reduced by one drop a week until it reaches one drop day. The drops discontinued within 5- 6 weeks. Complications are

elevation of IOP, cataract and uncommon complications are secondary corneal infection, cornea melting.

Periocular steroids

Periocular steroids are given as first line therapy to control inflammation and macular edema in unilateral posterior uveitis, to supplement systemic therapy or when systemic steroids are contraindicated in bilateral posterior uveitis. It delivers a therapeutic dose of medication close to site of inflammation. Triamcinolone acetonide (40mg) or methylprednisolone acetate (40-80mg) are most commonly used drugs.

Periocular injections can be given through transseptal or subtenon (Nozik technique) approach either in superotemporal (preferred) or inferotemporal quadrant. Patient is instructed to look down and nasally, after anesthesia is applied with a cotton swab soaked in proparacaine , needle is placed bevel down against the sclera and advanced through the conjunctiva and tenon capsule using side to side movement upto the hub,then drug is injected into the subtenon space.

Complications are ptosis, globe perforation, periorbital or retrobulbar hemorrhage, orbital fat atrophy, skin discoloration

andelevation of IOP for long time. Periocular injections are contraindicated in infectious posterior uveitis.

Systemic corticosteroids

Systemic steroids are used in vision threatening posterior uveitis, panuveitis or when systemic disease also requires therapy. Prednisolone is the most commonly used drug. Start with a large dose and then reduce. The starting dose of prednisolone 1-2mg/kg/ day given in a single morning dose after breakfast. A high level is maintained until a clinical effect is seen followed by a slow taper over every 1-2 weeks until the disease is quiescent. The lowest possible dose that will control inflammation and minimize side effects is desired. This dose should be 5-10mg/day. If dose greater than this is required or treatment required for longer than 3 months, steroid sparing immunomodulator therapy is indicated.

In explosive onset of severe non infectious posterior uveitis or panuveitis intravenous high dose pulse methylprednisolone (1g/day infused over 1 hour) therapy may be administered for 3 days followed by a gradual taper of oral prednisolone.

Side effects of short term therapy includes dyspepsia, mental changes, electrolyte imbalance and aseptic necrosis of the head of femur. Side effects of long term therapy includes cushingoid state, osteoporosis, reactivation of infections, cataract and exacerbation of diabetes and myopathy.

Contraindications are poorly controlled diabetes, peptic ulcer, osteoporosis, active systemic infection, psychosis on previous exposure to steroids.

Intravitreal administration

1) Intravitreal injections

2) Intravitreal implants

Intravitreal injection of preservative free triamcinolone acetonide through single trans pars plana route, 4mg/0.1ml given for recalcitrant uveitic cystoid macular edema and choroidal neovascularization. It produces sustained improvement in visual acuity for 3-6 months.

Intravitreal sustained release implants (fluocinolone acetonide 0.59mg, dexamethasone-ozurdex 350/700 microgram) are given for non infectious posterior uveitis. Early post operative complications are

endophthalmitis, wound leak, vitreous detachment and retinal detachment.Cataract and secondary glaucoma are the late common complications[2].

Immunomodulators

Antimetabolites, inhibitors of T cell signaling, alkylating agents and biological response modifiers are the commonly used immunomodulators.

Indications for immunomodulators in posterior uveitis are

- Severe vision threatening posterior uveitis
- Patients resistant to or cannot tolerate corticosteroids,
- When corticosteroids are contraindicated due to systemic problems
- Patient requires chronic corticosteroid therapy (longer than 3 months) at doses greater than 5-10 mg/day
- Chronic topical corticosteroid dependence and those who requiring multiple periocular corticosteroid injections.

Corticosteroids are the mainstay of initial therapy, but early use of immunomodulator therapy is indicated in certain specific uveitis (Serpiginous choroiditis, VKH, Bechets disease and sympatheticophthalmia) to improve the long term prognosis and tovisual morbidity. Before initiating the therapy, infections, hepatic and hematologic contraindications should be ruled out.

Therapeutic effect of immunomodulators will start only after several weeks. So patients should be maintainedon corticosteroids until the immunomodulators begin to take effect, at that timecorticosteroid dose gradually tapered.

Immunomodulatory drugs in posterior uveitis

Medications	Dosage	Complications
Antimetabolites		
Methotrexate	7.5-25 microgram/week	Hepatotoxicity, GI upset
Azathioprine	100-250mg/day	Hepatotoxicity, GI upset
Mycophenalate mofetil	1-3 g/day	GI ulceration, diarrhea
Inhibitors of T cell signaling		

Cyclosporine	2.5-5mg/kg/day	Nephrotoxicity,hypertension
Tacrolimus	0.1-0.2mg/kg/day	Nephrotoxicity,hypertension
Sirolimus	6mg iv loading dose, 4mg/day iv increased by 2mg increments	GI upset
Alkylating agents		
Cyclophosphamide	1-2mg/day	Hemorrhagic cystitis, sterility
Chlorambucil	2-12mg/day	Hemorrhagic cystitis, sterility
Biological response modifiers		
Infliximab	3mg/kg iv 0,2,6 weeks then once in 6-8weeks	Infusion reactions, infections, malignancy
Adalimumab	40mg/week	Headache, GI upset
Daclizumab	1mg/kg iv once in two weeks 5 doses	
Rituximab	Two doses 1g iv two weeks apart	Late onset neutropenia

Complications include hepatic and renal toxicity, bone marrow suppression and increased susceptibility to infections. Patients should be monitored with complete blood count, liver and renal function test

regularly. These agents are teratogenic, so patients should be advised to avoid pregnancy during treatment.

Treatment of Infectious posterior uveitis

Infectious posterior uveitis is treated with systemic antimicrobial drugs and systemic corticosteroids. Antibiotics, antiviral drugs, antifungal drugs, anti parasitic agents are the antimicrobial drugs used in posterior uveitis. The choice of antimicrobials depends on the etiology of posterior uveitis.

Treatment of sequelae and complications

Secondary glaucoma is treated with antiglaucoma medications. Topical corticosteroids and aqueous suppressants are administered topically and acetazolamide is given systemically. Pilocarpine and prostaglandin analogues are contraindicated in uveitic glaucoma.

Cataract are removed surgically after uveitis is quiescent for atleast 2-3 months. Cystoid macular edema is treated with meticulous control of intraocular inflammation with corticosteroids, immunomodulator drugs and periocular steroids.

Proliferative retinopathy is treated with laser photocoagulation. Permanent vitreous opacification affecting vision is treated with 3– port pars plana vitrectomy. Rhegmatogenous retinal detachment is treated with scleral buckling with cryoretinopexy, pars plana vitrectomy and endolaser with silicone oil tamponade or combined scleral buckling and pars plana vitrectomy. But the prognosis is poor . Retinal and Choroidal neovascularization are treated with control of inflammation using corticosteroids and immunomodulators, focal laser photocoagulation and intravitreal injectionof bevacizumab.

In spite of all treatment modalities 10% patients will develop permanent loss of vision. More so the patients with systemic association of diseases, treatment of posterior uveitis and restoration of vision is a challenge to all Ophthalmologists.

REVIEW OF LITERATURE

- Ocular toxoplasmosis is one of the most common type of Posterior uveitis of infectious origin comprising 30- 50% of cases. Pyrimethamine and sulfadiazine with corticosteroids is the classical chemotherapy most widely used in the treatment for ocular toxoplasmosis. An alternative treatment with Trimethoprim/sulfamethoxazole plus oral prednisolone showed to have similar efficacy to classical therapy (de-la-Torre A et al Therapy for ocular toxoplasmosis. Ocul Immunol Inflamm. 2011;19:314–320)[8.]

- In a study conducted by Anna Elias et a, the mean age presentation of VKH Syndrome was 37.26 years. Majority of patients had exudative retinal detachment, followed by optic dis edema and anterior uveitis. Fundus fluorescein angiography (FFA)showed multiple pinpoint hyperfluorescent leaks at the level of the retinal pigment epithelium in all patients. All patients were treated with systemic steroids. 17.39% patients were also treated with immunosuppressants. 5.26% patients developed subretinal fibrosis and 5.26% patients developed secondary

glaucoma 89.47% had a final visual acuity better than 6/12.The eyes with secondary glaucoma responded to topical anti-glaucoma meditations.

- The more common manifestation of ocular tuberculosis is chronic granulomatous iridocyclitis with multifocal posterior choroiditis. Choroidal granuloma is an atypical presentation for ocular tuberculosis and is less commonly reported.Patients are treated with category I ATT for total 6 months duration and Choroidal nodular lesion will resolve completely after 8 weeks of starting Anti-tubercular chemotherapy, with BCVA improving to 20/20. (Laurent J et al Arch Ophthalmol. 2005;123:864-866)[19].

- The most frequent clinical presentation of Eales' disease is a sudden painless loss of vision because of vitreous hemorrhage. Corticosteroids remain the mainstay of the treatment in the active perivasculitis stage of Eales' disease. Laser photocoagulation is the mainstay of treatment in the proliferative stage of Eales' disease. Fluorescein angiography helps in monitoring the response to treatment. (Abu El-Asrar and Al-Kharashi Br J Ophthalmol 86:1248-1251)[23].

- Serpiginous choroidopathy occur typically at the disc and spread in a helicoid pattern along the major vascular arcades towards the macula.Combination azathioprine and systemic corticosteroids is a safe and acceptable treatment . Recurrences are common and manifest as yellow-grey extensions at the level of the choriocapillaris, contiguous or as satellites to existing areas of chorioretinal atrophy . Visual disability may result directly from retinal lesions affecting the central macula, or from secondary choroidal neovascularization (Jampol LM et al *Am J Ophthalmol 88:683–689).*

- CMV retinitis is one of the most common complications of AIDS patients, it can affect 6 to 38% of patients and may be bilateral in 30 to 50% with up to 25% of the affected patients possibly losing their vision (Holland GN, et al . *American Journal of Ophthalmology*, 114: 86-95).

- Ocular toxocariasis is relatively uncommon,occurring in1% of uveitis. Clinical presentation is unilateral (90.9%). Ocular Toxocarasis is characterized by a granuloma in the peripheral retina in 50% of cases, granuloma in the macula in 25% of cases, and severe vitreous inflammation mimicking endophthalmitis in

25% cases. The primary causes of poor vision are cystoid macular edema (47.4%), and traction retinal detachment (36.8%). The mainstay of treatment of Toxocariasis is corticosteroids to reduce inflammatory responses (Smith H et al Trends Parasitol 25: 182–188. doi: 10.1016/j.pt.2009.01.006) .

- Ocular manifestations in SLE may be vision threatening and is indicator of active systemic disease. The mainstay of treatment is systemic corticosteroids and immunosuppressive drugs.Proliferative retinopathy is treated with panretinal photocoagulation Giorgi D et al.

PART II

AIM OF THE STUDY

To analyse the posterior uveitis with reference to age and sex incidence, laterality, etiology, chronicity, severity, clinical presentation, sequelae, complication and treatment modalities.

Primary objective

- To evaluate the various etiological factors and predisposing factors leading on to posterior uveitis.
- To assess the clinical presentation in different types of posterior uveitis.
- To diagnose and start treatment after systematic evaluation

Secondary objective

- To assess visual outcome after treatment of Posterior Uveitis since Posterior uveitis treatment is a challenge to all Ophthalmologists.

MATERIALS AND METHODS

The study design was a prospective study of 30 patients with age range of 20-45 years with posterior uveitis, conducted in Regional Institute of Ophthalmology and Ophthalmic Eye Hospital during August 2013 to August 2014 for a period of 12 months.

Main criteria for diagnosis

1) Patients having whitish retinal opacities with indistinct borders (focal/multifocal/diffuse) and vitritis suggestive of retinitis

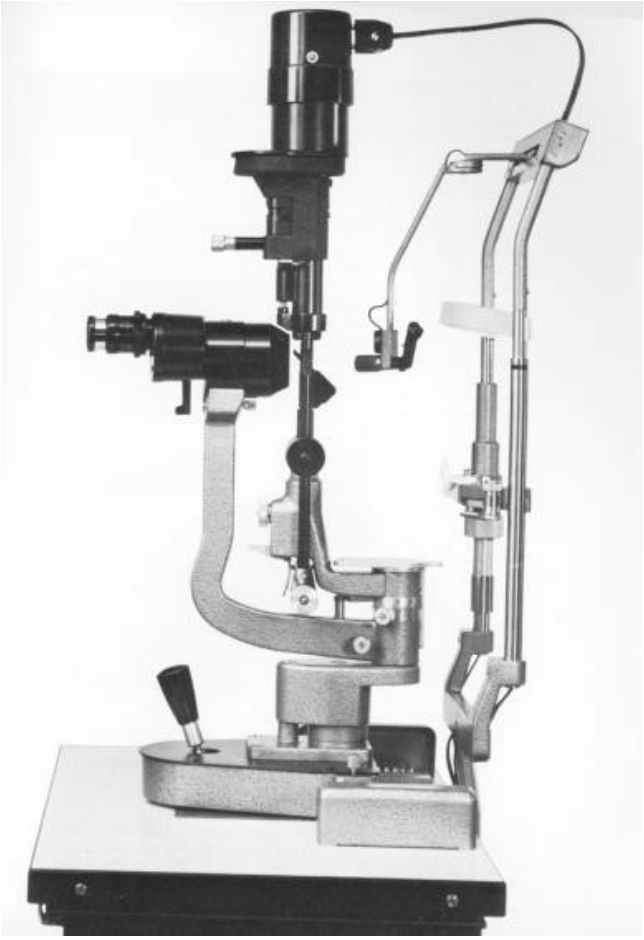
2) Patients having round yellowish nodule in the fundus (focal/multifocal/diffuse) suggestive of choroiditis

3) Patients having yellow or grey white, patchy perivascular cuffing suggestive of active vasculitis

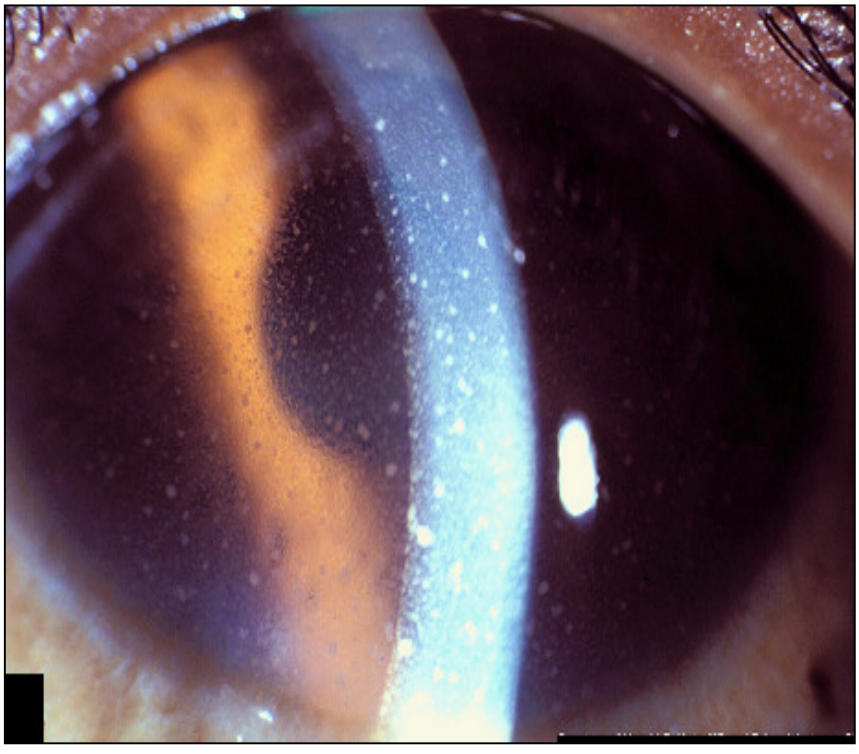
4) Patients having papillitis with multiple serous detachment suggestive of VKH syndrome

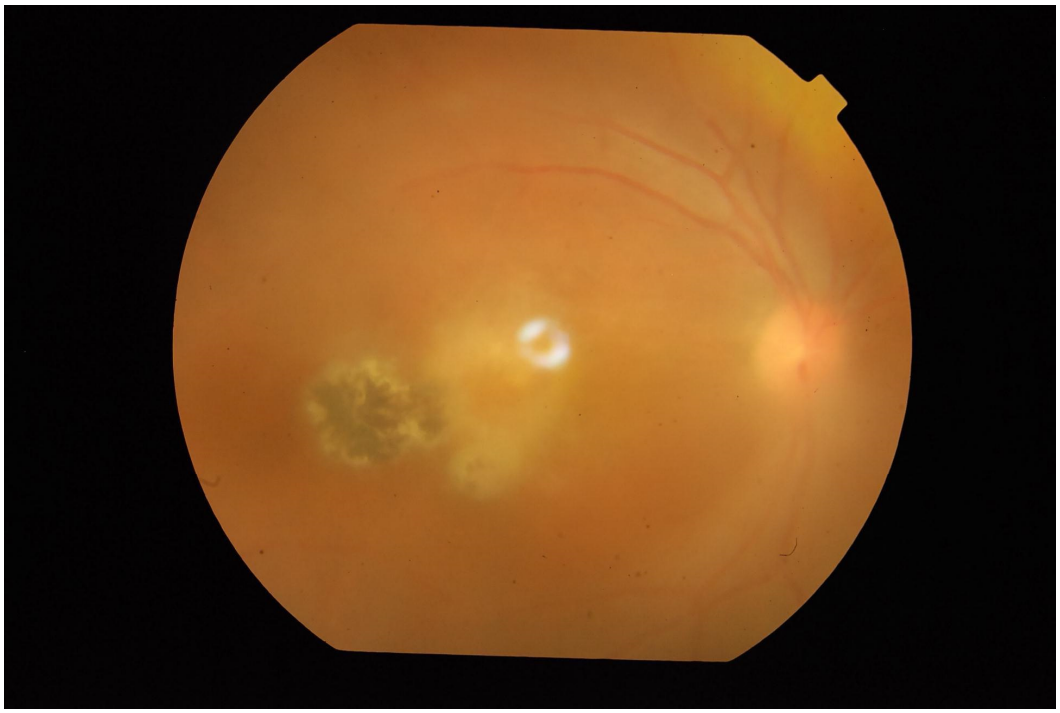
5) Patients having peripheral tubercles

SLIT LAMP EXAMINATION



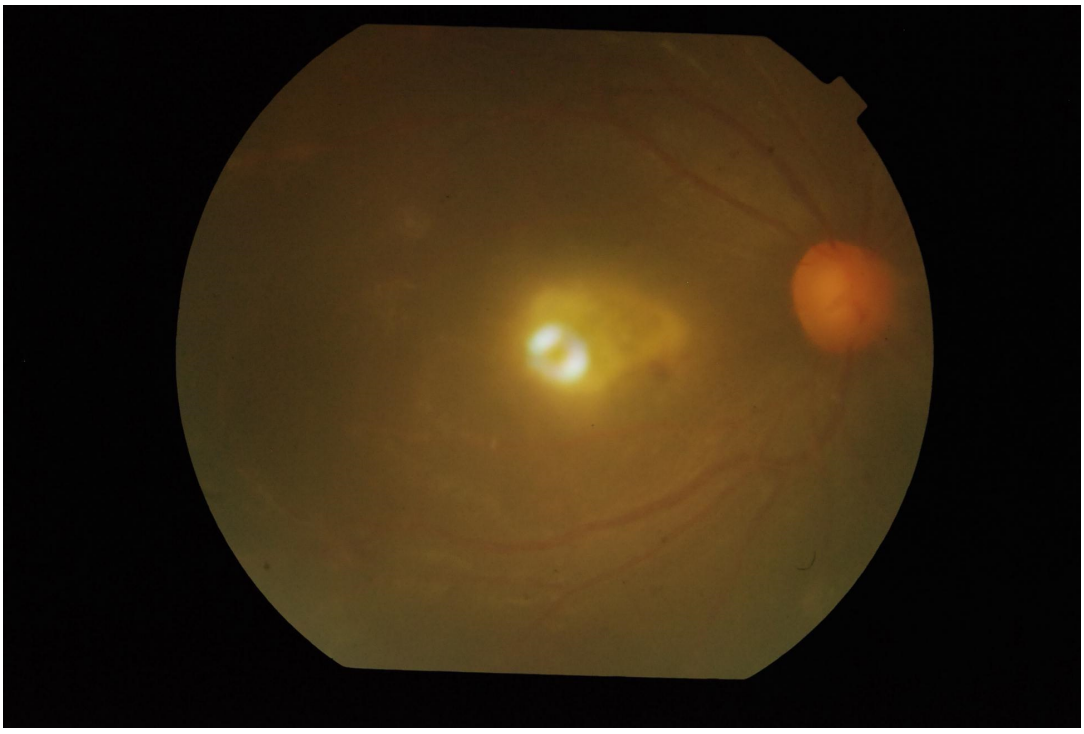
KERATIC PRECIPITATES





ACTIVE CHOROIDITIS ADJACENT TO PIGMENTED CHORIORETINAL SCAR

HEAD LIGHT IN THE FOG APPEARANCE



Exclusion criteria

1) Post surgical cases

2) Post trauma cases

3) Intermediate uveitis (predominant vitritis without active chorioretinal lesion)

4) Predominant anterior segment involvement without active chorioretinal lesion)

5) Patients who lost follow up.

All these patients were questioned about thepresenting compliants (floaters, defective vision), history of contact with pets, tuberculosis, joint pain and focal sepsis.

Systemic examination of cardiac, pulmonary, gastrointestinal, central nervous system and musculoskeletal system was done.

Complete ocular examination including visual acuity using Snellen’s acuity chart, slit lamp examination of the anterior segment, lens and vitreous, posterior segment examination with direct and indirect

ophthalmoscopy, slit lamp biomicroscopy using 90 diopter lens, intra ocular pressure measurement were done.

All these patients were subjected to a battery of investigations including total count, differential count, erythrocyte sedimentation rate, chest x ray PA view, mantoux test and blood sugar. Further investigations depends on probable etiology of posterior uveitis. TORCH screening and Quantiferon gold were done in infectious cases.

Fundus fluorescein angiography was done in non infectious cases. ANA, dsDNA antibody was done in suspected cases of systemic lupus erythematosus. Optical coherent tomography was done in choroidal neovascularization. CD4 count was done in suspected cytomegalovirus infection. Liver function tests and renal function tests were done in patients who required immunosuppressive therapy.

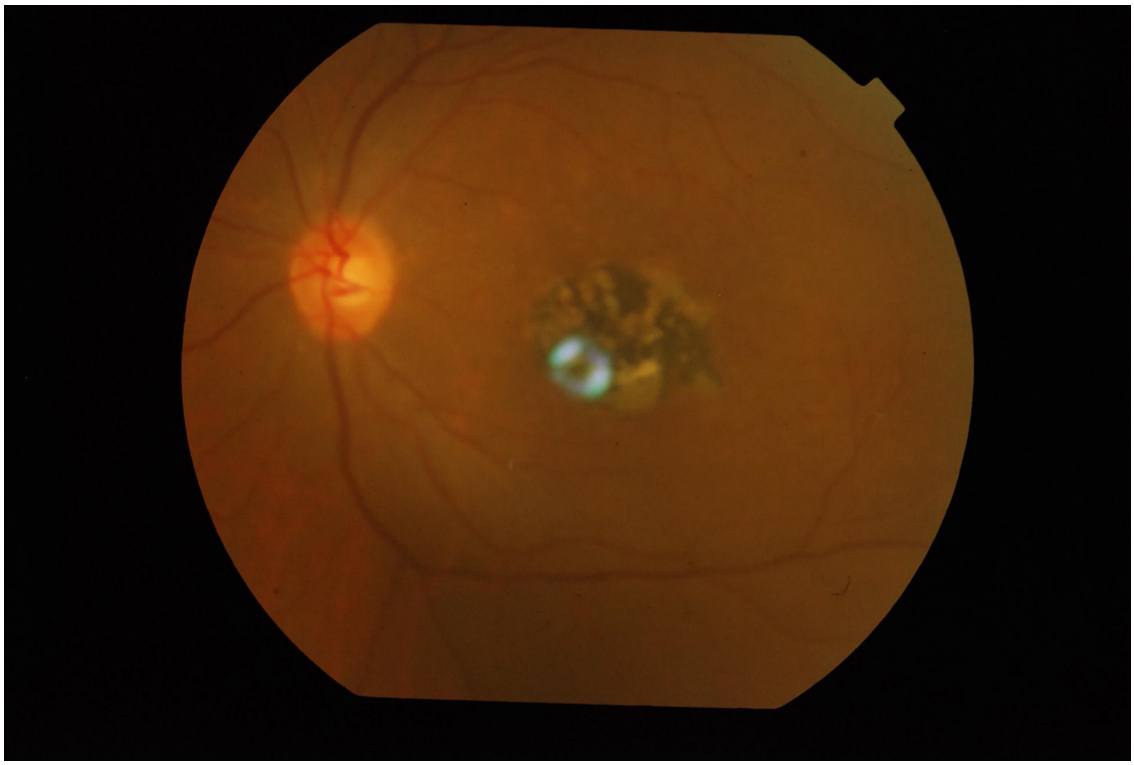
Patients were treated with topical and systemic corticosteroid therapy in non-infectious posterior uveitis, systemic antimicrobials, topical and systemic corticosteroids in infectious posterior uveitis following which dose will be tapered over a period of 2 months. Immunosuppressive therapy was given in patients not responding to corticosteroids and those who required prolonged corticosteroid therapy.

Proliferative retinopathy was treated with panretinal photocoagulation. Intravitreal injection bevacizumab was given for choroidal neovascularization. Post inflammatory glaucoma was managed with topical antiglaucoma medications.

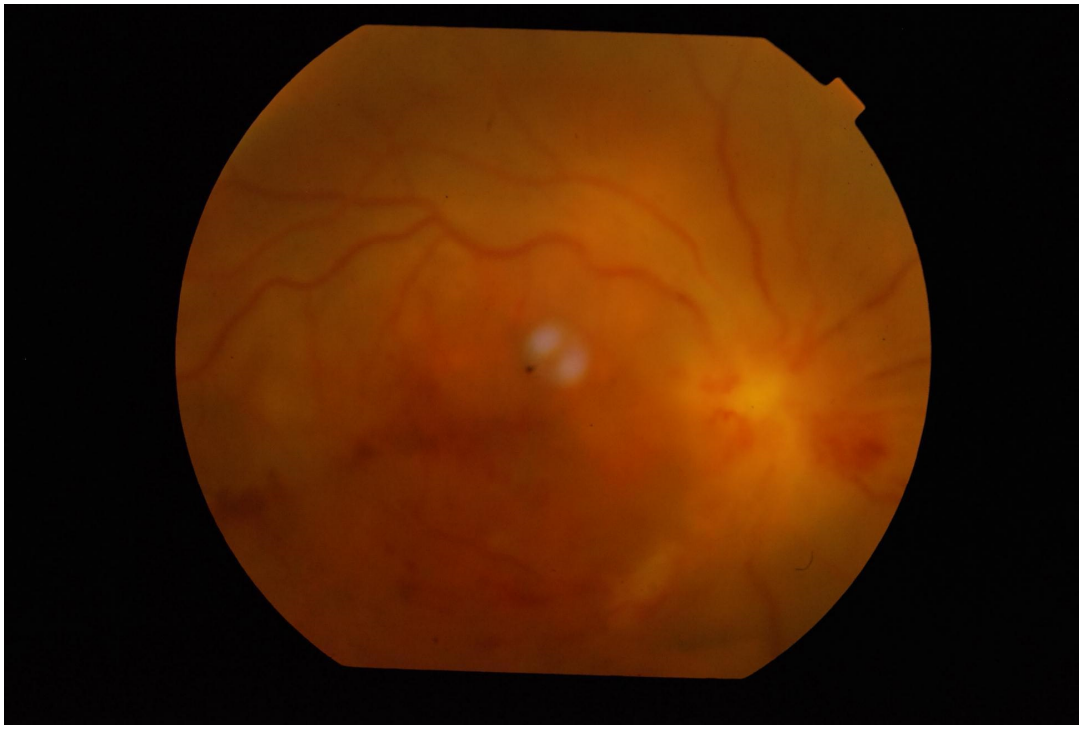
All patients were followed up every week for 4 weeks, every two weeks for one month then every month for 4 months, totally of 6 months.

Improvement in visual acuity, intra ocular pressure, resolution of vitritis and choroidal leisons were assessed during follw up.

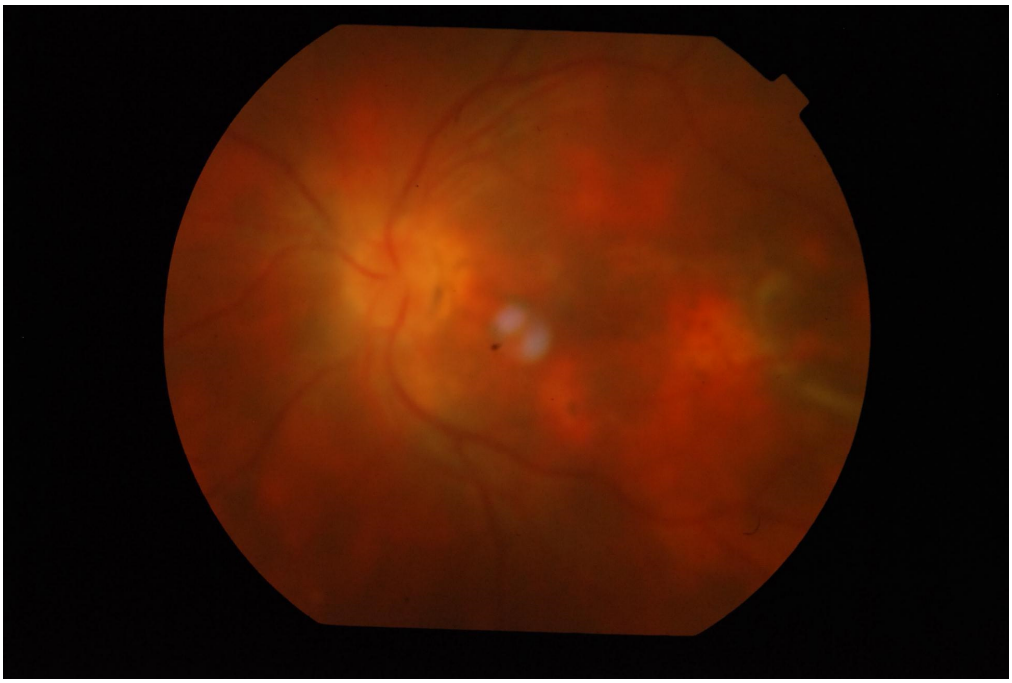
HEALED CHOROIDITIS



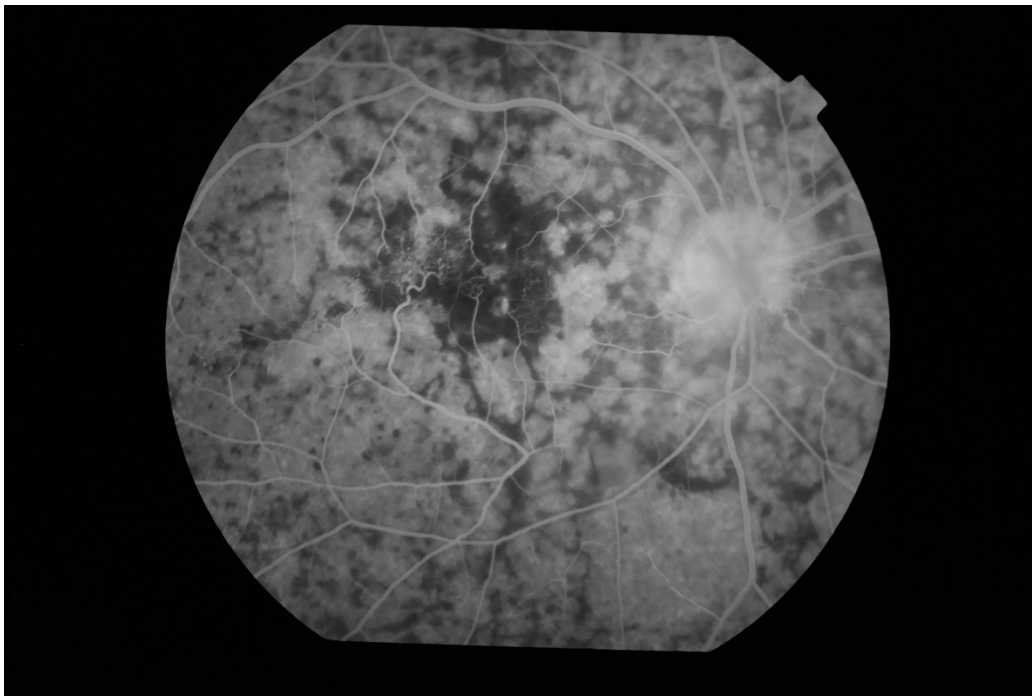
ACUTE VKH SYNDROME



CHRONIC VKH SYNDROME



FFA WINDOW DEFECTS IN VKH



RESULTS AND ANALYSIS

Table 1 : Age Distribution

Age Group	Cases	
	No	**%**
21 25 yrs	6	20
26 – 30 yrs	8	26.7
31 – 35 yrs	7	23.3
36 – 40 yrs	6	20
41 – 45 yrs	3	10.0
Total	**30**	**100**
Range	21 – 45 yrs	
Mean	32.2 yrs	
SD	6.3 yrs	

In our study maximum number of cases posterior uveitis were found to be within the age groups of 26-30 years and 31-35 years comprising 26.7% and 23.3% respectively.

Table 2 : Sex Distribution

Sex	**Cases**	
	No	**%**
Male	14	46.7
Female	16	53.3
Total	**30**	**100**

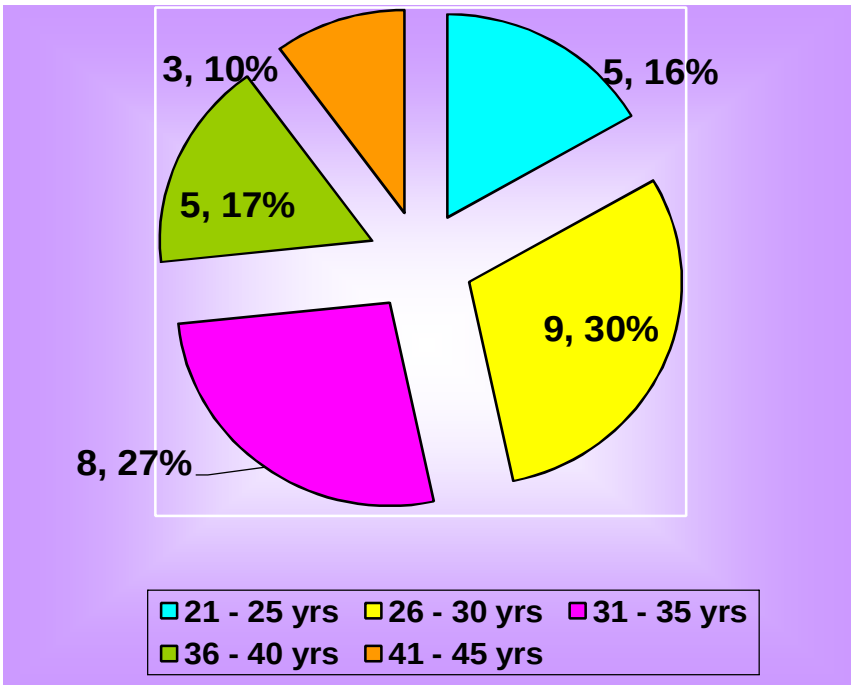
Out of cases in our study 14 cases were males (46.7%), 16 cases were females (53.3%). Not much of sexual predilection towards any group in our study.

Table 3 : Laterality

Laterality	**Cases**	
	No	**%**
Unilateral	15	50
Bilateral	15	50
Total	**30**	**100**

Regarding the laterality, out of 30 cases in our study 15 cases (50%) were unilateral and 15 cases were bilateral (50%). The laterality was equal in our study.

AGE DISTRIBUTION



SEX DISTRIBUTION

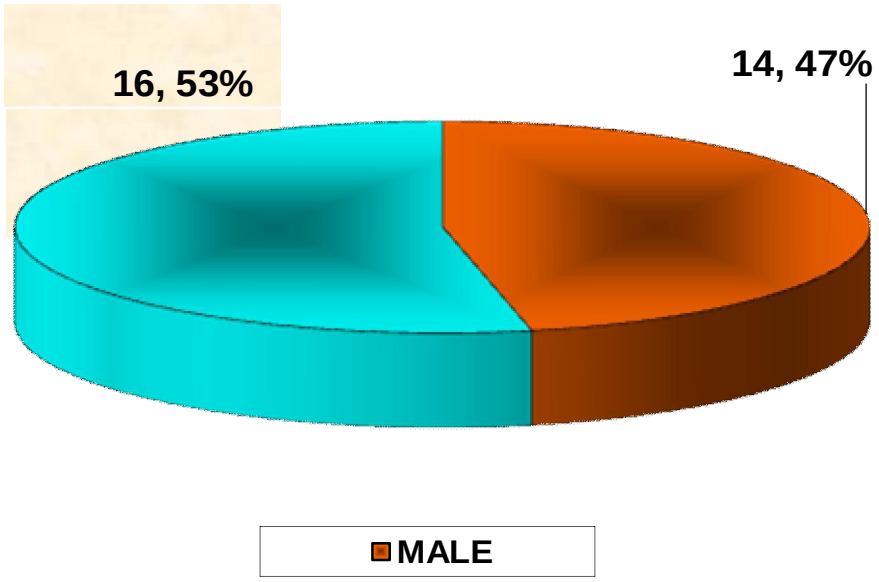


Table 4 : Duration and Onset

Duration	No of cases	Percentage
Acute	0	0
Acute recurrent	0	0
Chronic	30	100%

In our study out of 30 cases, all cases (100%) were of chronic duration of more than 6 weeks. There was no acute and acute recurrent cases.

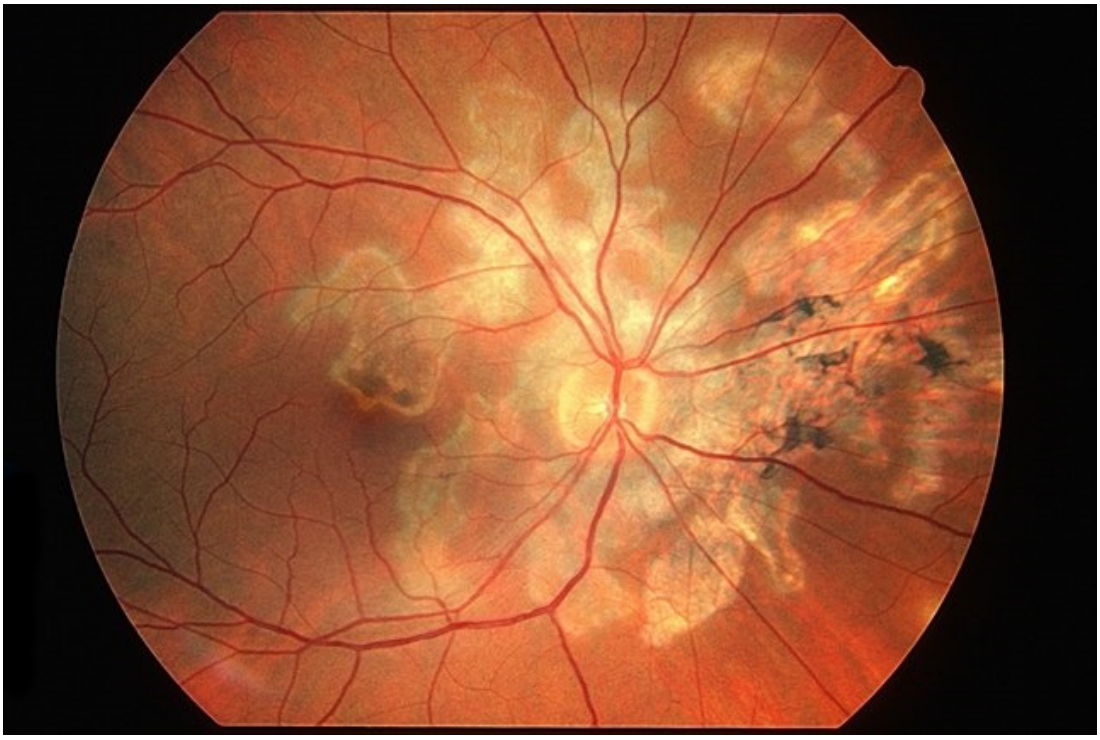
Table 5: Severity of posterior uveitis

Severity	No. of cases	Percentage
Mild	0	0%
Moderate	0	0%
Severe	30	100%

In our study out of 30 cases, all cases (100%) were of severe in nature.

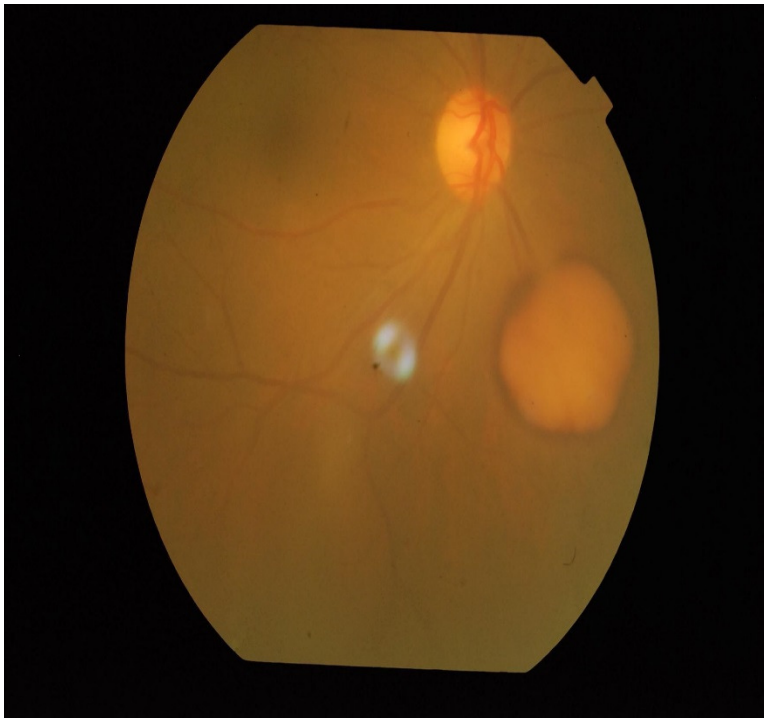
CMV RETINITIS

SERPIGINOUS CHOROIDOPATHY



CHOROIDAL TUBERCULOMA

BEFORE TREATMENT



AFTER TREATMENT

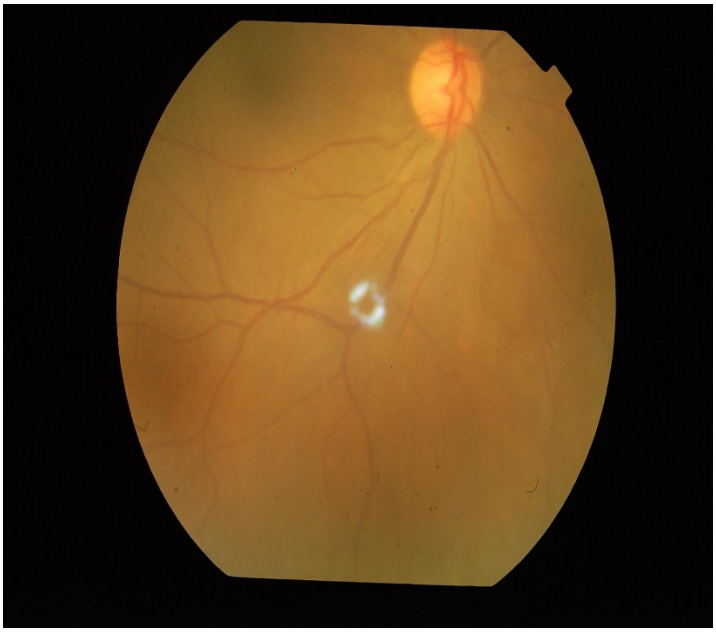
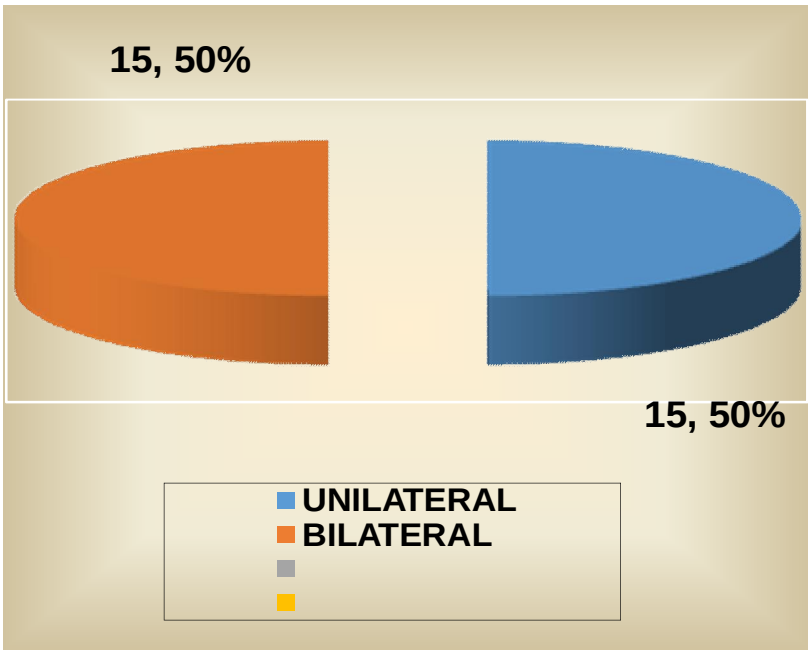


Table 6 : Aetiology of posterior uveitis

Aetiology	No of cases	Percentage
Infectious	18	60%
Non infectious	12	40%
Total	**30**	**100%**

In our study out of 30 cases, 18 cases (60%) were of infectious etiology and 12 cases (40%) non infectious etiology. Infectious etiology was more common than non infectious etiology in our study.

LATERALITY



AETIOLOGY OF POSTERIOR UVEITIS

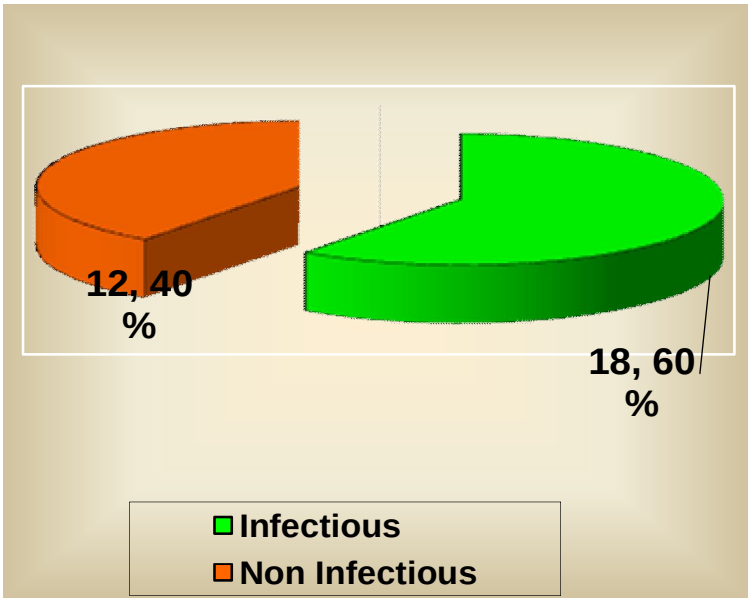
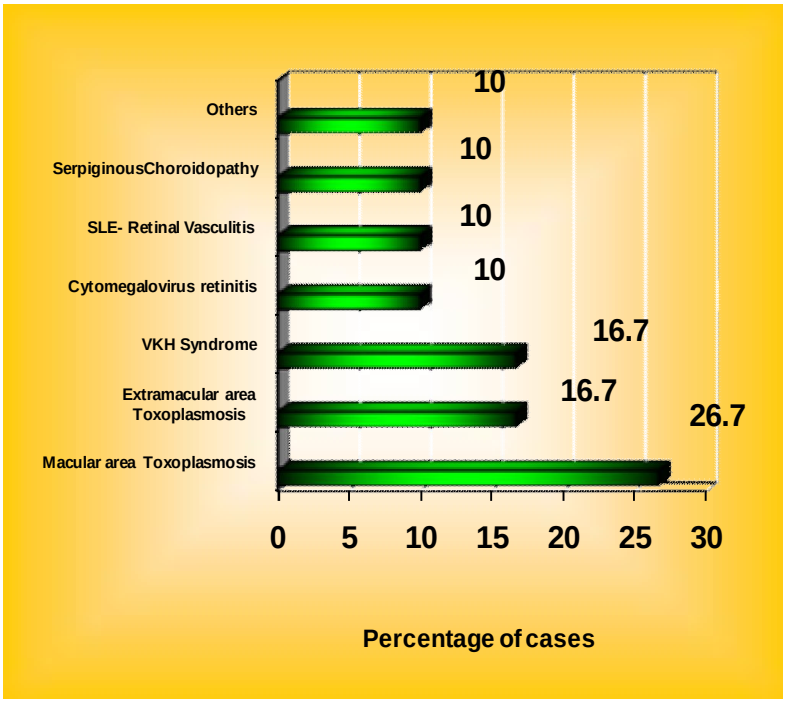


Table 7 : Aetiological Diagnosis

Aetiology	Cases	
	No	**%**
Toxoplasmosis		
a) Macular area	8	26.7
b) Extramacular area	5	16.7
c) Total	13	43.3
VKH Syndrome	5	16.7
Cytomegalovirus retinitis	3	10.0
SLE- Retinal Vasculitis	3	10.0
Serpiginous Choroidopathy	3	10.0
Eale's disease	1	3.3
Tuberculosis	1	3.3
Toxocariasis	1	3.3
Total	**30**	**100**

In the aetiological analysis, 13 cases (43.3%) were of Toxoplasma etiology, 5 cases (16.7%) of VKH Syndrome, 3 cases (10.0%) were of serpiginous choroidopathy, 3 cases (10.0%) were of cytomegalovirus retinitis, 3 cases (10.0%) were of SLE retinal vasculitis. Eale's disease was found in one case (3.3%), one case (3.3%) had Tuberculous etiology and Toxocariasis was found in one case (3.3%).

AETIOLOGICAL DIAGNOSIS



ANATOMICAL CLASSIFICATION

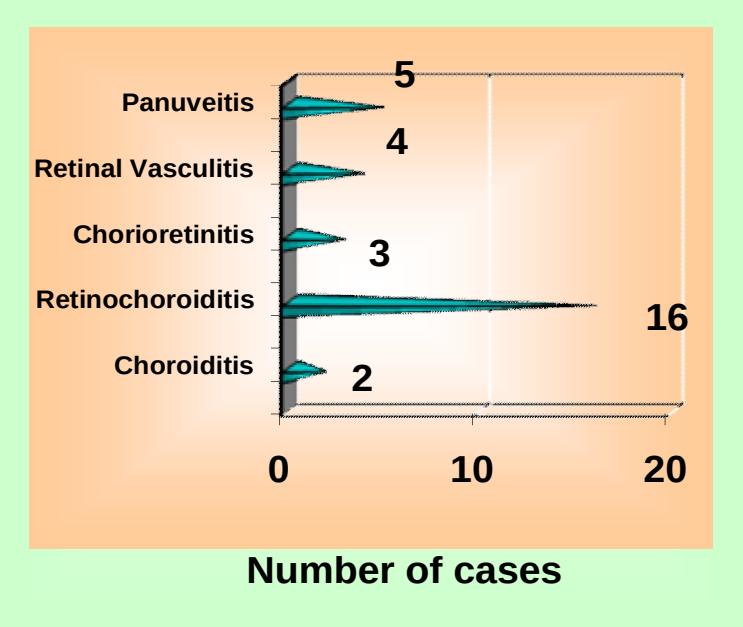


Table 8 :Anatomical classification

Anatomical Classification	Cases	
	No	**%**
Choroiditis	2	6.7
Retinochoroiditis	16	53.3
Chorioretinitis	3	10.0
Retinal Vasculitis	4	13.3
Panuveitis	5	16.7
Total	**30**	**100**

Based on anatomical classification of Posterior uveitis in our study, 16 cases (53.3%) of cases were Retinochoroiditis, 4 cases (13.3%) were Retinal vasculitis, 3 cases (10.0%) were Chorioretinitis, 2 cases (6.7%) were Choroiditis and 5 cases (16.7%) were found to be Panuveitis.

Table 9 : Aetiological analysis based Anatomical classification.

Aetiology	Choroditis		Retina choroditis		Chorio Retinitis		Retinal Vasculitis		Panuveitis	
	No	%	No	%	No	%	No	%	No	%
Toxoplasmosis (13)	-	-	13	81.25	-	-	-	-	-	-
VKH Syndrome (5)	-	-	-	-	-	-	-	-	5	100
Serpiginous Choroidopathy (3)	-	-	-	-	3	100	-	-	-	-
SLE-Retinal Vasculitis (3)	-	-	-	-	-	-	3	75	-	-
Cytomegalovirus retinitis (3)	-	-	3	18.75	-	-	-	-	-	-
Eale's disease (1)	-	-	-	-	-	-	1	25	-	-
Tuberculosis (1)	1	50	-	-	-	-	-	-	-	-
Toxocariasis (1)	1	50	-	-	-	-	-	-	-	-

In case of Choroiditis only 2 cases were present in our study in that one was Tuberculosis another was Toxocariasis. In Retinochoroiditis 81.25% of cases were Toxoplasmosis and 18.75% of cases were Cytomegalovirus infection. In retinal vasculitis 75% were

due to systemic lupus erythematosus and 25% was due to Eale’s disease. Panuveitis is mainly due to VKH Syndrome (100%).

In our study choroiditis and chorioretinitis were of infectious origin. Retinal vasculitis was due to SLE and Eales disease.

Table 10 :Age incidence based on anatomical classification

Age	No of cases	Choroditis		Retina choroditis		Chorio Retinitis		Retinal Vasculitis		Panuveitis	
		No	%	No	%	No	%	No	%	No	%
21 - 25	6	1	16.7	4	66.7	-	-	1	16.7	-	-
26 - 30	8	-	-	4	50	-	-	2	25	2	25
31 - 35	7	--	-	4	57.1	-	-	1	14.3	2	28.6
36 - 40	6	-	-	3	50	2	33.3	-	-	1	16.7
41 - 45	3	1	33.3	1	33.3	1	33.3	-	-	-	-
Total	30	2	6.7	16	53.3	3	10.0	4	13.3	5	16.7

In our study maximum number of cases posterior uveitis were found to be within the age groups of 26-30 years and 31-35 years comprising 26.7% and 23.3% respectively. Age group affected in our study was between 21- 45 years of age.

Table 11 :Sex incidence based on anatomical classification.

Sex	Choroditis		Retina choroditis		Chorio Retinitis		Retinal Vasculitis		Panuveitis	
	No	%	No	%	No	%	No	%	No	%
Male	1	50	8	50	3	100	1	25	2	40
Female	1	50	8	50	-	-	3	75	3	60

In Choroiditis one case(50%) was male and one case (50%) was female. In Retinochoroiditis 8 cases (50%) were males and 8 cases (50%) were females. In Chorioretinitis all cases (100%) were males. In Retinal vasculitis one case (25%) was male and 3 cases (75%) were females. In Panuveitis 2cases (40%) and 3 cases (60%) were females.

Table 12 :Laterality based on anatomic classification

Laterality	Choroditis		Retina choroditis		Chorio Retinitis		Retinal Vasculitis		Pan uveitis	
	No	**%**	**No**	**%**	**No**	**%**	**No**	**%**	**No**	**%**
Unilateral	2	100	13	81.25	-	-	-	-	-	-
Bilateral	-	-	3	18.75	3	100	4	100	5	100

Based on anatomical classification, all cases of Choroiditis were unilateral. In Retinochoroiditis 13 cases (81.25%) were unilateral and 3 cases (18.75%) were bilateral. In Chorioretinitis all cases were bilateral. In Retinal vasculitis all cases were bilateral. All cases of Panuveitis were bilateral. Bilateral retinochoroiditis were due to CMV retinitis.

Table 13 : Vitreous

Vitreous	No of cases	Percentage
Normal	7	23.3
Grade I vitreous haze	8	26.6
Grade II vitreous haze	1	3.3
Grade III vitreous haze	13	43.3
Grade IV vitreous haze	0	0
Vitreous hemorrhage	1	3.3
Total	**30**	**100**

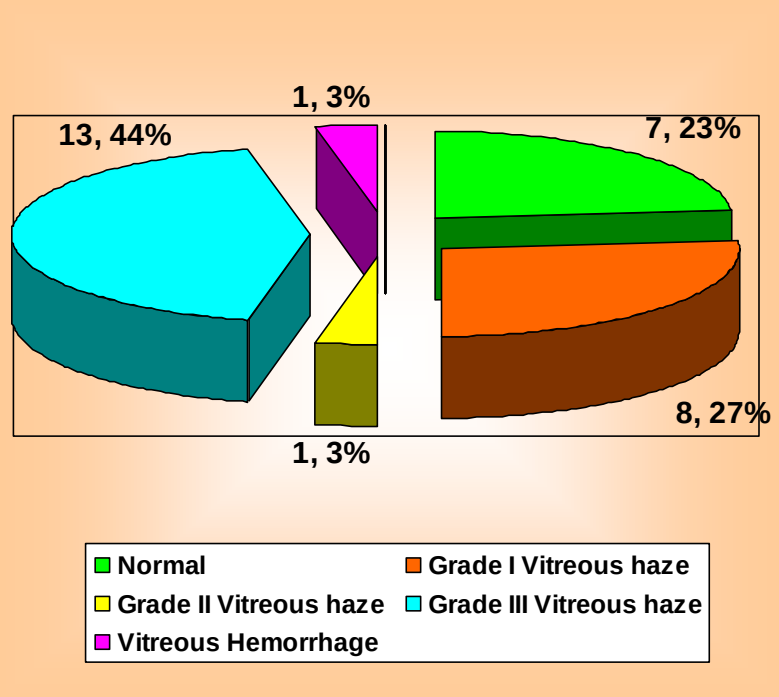
In our study out of 30 cases, grade III vitritis was seen in 13 cases (43.3%), grade I vitritis was seen in 8 cases (26.6%),grade II vitritis was seen in 1 case (3.3%). Vitreous hemorrhage was seen in one case (3.3%) and vitreous was normal in 7 cases (23.3%). Majority of cases in our study were presented with grade III vitritis.

Table 14 : Investigations

Investigations	Cases	
	No	**%**
TORCH Screening, TC, DC, ESR,	17	56.7
FFA, RFT, LFT	12	40.0
Chest X Ray, Mantoux test	1	3.3
Quantiferon Gold	1	3.3
OCT	1	3.3
CD4 Count	3	10.0
ANA, anti ds DNA Antibody, RF, cANCA, pANCA	3	10.0

In our study most common investigation required was TORCH screening,TC, DC and ESR for 17 cases (56.7%), followed by Fundus Fluorescein Angiography, RFT and LFT for 12 cases (40%), CD4 count for 3 cases (10%). Other investigations, Chest X Ray, Mantoux test, ANA, anti ds DNA Antibody, RF, cANCA, pANCA, Quantiferon Gold and OCT were required in one case (3.3%).

VITREOUS HAZE



INVESTIGATIONS

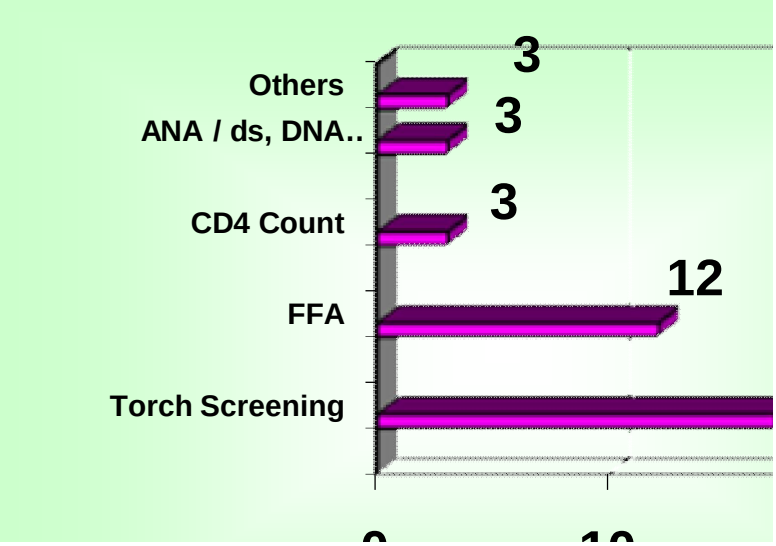


Table 15 : Treatment

Treatment	Cases	
	No	**%**
Systemic corticosteroids	27	90
Topical mydriatics and cycloplegics	17	56.7
Topical corticosteroids	17	56.7
Anti Toxoplasma agents	13	43.3
Immunosuppressive drugs	11	36.66
Anti cytomegalovirus drugs	3	10.0
Panretinal photocoagulation	3	10.0
Anti tubercular drugs	1	3.3
Intra vitreal injection Bevacizumab	1	3.3
Anti glaucoma medications	1	3.3

In our study most common drugs used for treatment were systemic corticosteroids, required in 27 cases (90%), followed by topical corticosteroids, topical mydriatics and cycloplegics in 17 cases(56.7%). Anti Toxoplasma agents were needed in 13 cases (43.3%) and

Immunosuppressive drugs were needed in 11 cases (36.66%). Anti cytomegalovirus drugs and panretinal photocoagulation were given for 3 cases (10%).

Other treatments like Anti tubercular drugs, Intra vitreal injection Bevacizumab and Anti glaucoma medications were given in one case (3.3%).

Table 16 : Complications

Complications	Cases	
	No	**%**
Cataract	4	13.3
Glaucoma	1	3.3
Proliferative Retinopathy	3	10.0
Retinal Detachment	1	3.3
Subretinal Fibrosis	1	3.3
Choroidal Neovascularization	1	3.3

In our study, out of 30 cases of posterior uveitis, cataract was seen in 4 cases(13.3%), proliferative retinopathy in 3 cases(10%), subretinal fibrosis and choroidal neovascularization were seen in one case (3.3%). Post inflammatory glaucoma was seen in one case (3.3%).

COMPLICATIONS

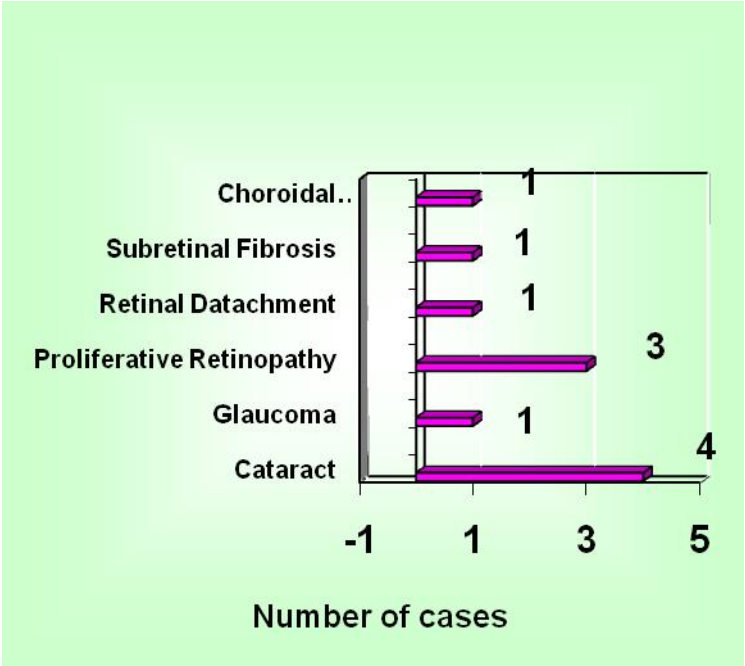


Table 17 : Visual outcome

Visual improvement	No. of cases	Percentage
6/6	3	10%
6/12 – 6/9	5	16.66%
4/60 – 6/36	20	66.66%
No improvement	2	6.66%

In our study of 30 cases, even with early diagnosis and prompt treatment only in 3 cases (10%) vision improved to 6/6 and 5 cases (16.66%) improved to 6/12 to 6/9. In remaining 20 cases (66.66%) there was some visual improvement that is not very significant and in 2 cases (6.66%) there was no visual improvement.

Table 18 : Visual Outcome based on aetiological diagnosis

Disease	Disease Contained	No of cases	Visual Outcome		Remarks	Visual Improvement
			Pre Treatment	Post Treatment		
Toxoplasmosis	Yes	5	6/36– 6/24	6/12 – 6/9	Extra Macular area	Improved
		8	1/2/60– 2/60	6/60– 6/36	Macular involvement	Improved
VKH Syndrome	Yes	3	HM – 6/36	6/12 – 6/6	-	Improved
		1	1/60	1/60	Choroidal neovascularization	Not Improved
		1	3/60	6/24	Subretional fibrosis	Improved
Cytomegalovirus retinitis	Yes	3	HM – 3/60	4/60 – 6/60	Macular involvement	Improved
Serpiginous Choroidopathy	Yes	3	4/60 – 6/36	6/36 – 6/12	Chorioretinal atrophy	Improved
SLE – retinal Vasculitis	Yes	3	1/60 – 4/60	6/60 – 6/36	Proliferative retinopathy	Improved
Eale's disease	Yes	1	HM	6/24	Proliferative Retinopathy	Improved
Tuberculosis	Yes	1	1/60	6/36	-	Improved
Toxocariasis	Yes	1	3/60	1/60	Tractional Retinal Detachment	Not Improved

In our study out of 13 cases of Toxoplasmosis, 5 cases were involved the extramacular area with good visual outcome , vision improved to 6/12 to 6/9. 8 cases were showed macular involvement with less improvement in vision in the range of 6/60 to6/36.

In VKH Syndrome, out of 5 cases 3 cases were improved vision significantly 6/9 to 6/6. One case had subretinal fibrosis in macular area with vision improving to 6/24. In one case was not improved due to choroidal neovascularization.

In cytomegalovirus infection there was only mild improvement in vision (4/60 to6/60) due to macular involvement. In serpiginous choroidopathy visual outcome was affected by chorioretinal atrophy in macular area, improved upto 6/36 to 6/12.

In Retinal vasculitis due to SLE (6/60 to 6/36) and in Eales disease (6/24)visual outcome was affected by proliferative retinopathy. In tuberculosis vision was improved to 6/36 and no complications.

In Toxocariasis vision was not improved (1/60) due to development of tractional retinal detachment.

Table:19 Cause for poor visual outcome

Cause	No. of cases	Percentage
Chorioratinal atrophy in macula	14	46.66%
Proliferative retinopathy	3	10%
CNVM	1	3.33%
Sub retinal fibrosis	1	3.33%
Tractional Retinal Detachment	1	3.33%

In our study, the most common cause for poor visual outcome is chorioretinal atrophy (46.66%) seen in 14 cases followed by proliferative retinopathy seen in 3 cases (10%). Others were CNVM, subretinal fibrosis and tractional retinal detachment seen in 3.33% of cases.

Table 20 : Sequelae and Complications

Pathology	**Remarks**	**No. of cases**	**percentage**
Structural damage	Chorioretinal atrophy	14	46.66%
Complication	CNVM, subretinal fibrosis, tractional retinal detachment, proliferative retinopathy	6	20%

In our study, poor visual outcome due to structural damage (chorioretinal atrophy) seen in 14 cases (46.66%) and due to complications were (20%)

In our study, Structural damage caused by the disease was more common than the complications.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square, ‘t’ value and 'p' values were calculated. ‘t’ test was used to test the significance of difference between quantitative variables and Yate’s and Fisher’s chi square tests for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

DISCUSSION

In our study of 30 cases of Posterior Uveitis, it was observed that posterior uveitis is more common in the age group of 26 to 35 years. Sex incidence is almost equal in males and females. Unilateral and bilateral cases are equal in ocurrence. Allof the cases were of chronic duration.

On analyzing the aetiology of posterior uveitis, in our study majority of cases were found to be due to toxoplasmosis (43.3%), followed by VKH syndrome (16.7%). Toxoplasmosis was the most common infectious etiology found in our study.

Anatomical classification

In our study majority of cases were Retinochoroiditis (53.3%), followed by Panuveitis (16.7%), Retinal vasculitis (13.3%), Chorioretinitis (10%) and Choroiditis(6.7%).

Aetiological analysis

In our study, aetiological analysis of posterior uveitis showed that majority of cases were of Toxoplasmosis(43.3%), followed by VKH syndrome.

Retinal vasculitis with Systemic Lupus Erythematosus were present in3 cases(females), 2 out of thatwere getting treatment at Rheumatology department,Government General Hospital (GGH) with systemic corticosteroids for the past 6 months with ANA anti ds DNA antibody positivity. One case was presented to our hospital with defective vision and Rheumatoogy workup done at rheumatology department GGH, found to be ANA and anti ds DNA antibody positivity and started on systemic corticosteroids.

3 cases were cytomegalovirus retinitis, out of that one case was on treatment with anti retroviral drugs for the past 2 months in ART centre with low CD4 count. Other 2 cases were presented to our institute with defective vision and referred to ART centre for evaluation found to be HIV positive with low CD4 levels and started on anti retroviral therapy.

Tuberculous etiology was found in one case with quantiferon gold positivity and sent to Thoracic medicine department at GGH for evaluation and treated with antituberculous drugs and systemic corticosteroids.

Clinical presentation

In our study, posterior uveitis is more common in age group of 26 – 35 years. Majority of cases of infectious posterior uveitis were unilateral and non infectious posterior uveitis and panuveitis cases were bilateral.

Complications

Since the main reason for poor visual outcomeis the structural damage caused by the disease process, there is no significant visual improvement even after the complete remission of the disease.

In our study cataract was present in 4 cases (13.3%), mainly in cases of VKH syndrome.3 cases were presented with proliferative retinopathy, 2 out of that were due to systemic lupus erythematosus with retinal vasculitis, one was Eales disease.

Post inflammatory glaucoma was present in one case of VKH syndrome. Subretinal fibrosis was present in one case and choroidal neovascularization was present in another case of VKH syndrome.

Retinal detachment was present in one case of toxocariasis.

Treatment

In our study the most common drugs used for treatment is systemic and topical corticosteroids followed by topical mydriatrics and cycloplegics and anti toxoplasma agents.

Since all cases of posterior uveitis are of chronic in nature, Immunomodulators are used in 11 cases (36.66%) as steroid sparing agents in non infectious posterior uveitis and retinal vasculitis.

In our study, toxoplasmosis cases were treated with anti toxoplasma agents and systemic corticosteroid. CMV retinitis patients were treated with anti CMV agents. Serpiginous choriodopathy, SLE with retinal vasculitis and Eales disease patients are treated with systemic corticosteroid agents.

VKH patients were treated with topical corticosteroids, mydriatics and cycloplegics, systemic corticosteroids and Immune modulators. Immuno modulator therapy was given in VKH syndrome in Serpiginous choroidopathy and SLE with retinal vasculitis.

Proliferative retinopathy cases were treated with Panretinal photocoagulation, secondary glaucoma was treated with anti glaucoma agents and choroidal neovascularization was treated with intra vitreal

bevacizumab.Since cataract was in early stage, without obstructing the visual axis they were not treated.

Visual outcome

In our study, due to early diagnosis and prompt treatment the disease was adequately controlled in all cases. But only 26.66% of patients obtained significant visual improvement. Other 66.66% of patients there is some visual improvement that is not very significant. The main reason for poor visual outcome was chorioretinal atrophy in the macula, the structural damage caused by the disease followed by the complications namely sub retinal fibrosis and proliferative retinopathy.

In 6.6% of patients, there was no visual improvement due to complications mainly choroidal neovascularization and tractional retinal detachment. In choroidal neovascularization, after treatment scarring was occurred but there was no visual improvement.

Out of 13 cases of Toxoplasmosis, 5 cases were involved the extramacular area with good visual outcome and 8 cases were showed macular involvement with less improvement in vision.

In VKH Syndrome, out of 5 cases 3 cases were improved vision significantly . One case had subretinal fibrosis in macular area with

vision improving to 6/24. In one case vision was not improved due to choroidal neovascularization.

In cytomegalovirus infection there was only mild improvement in vision due to macular involvement. In serpiginous choroidopathy visual outcome was affected by chorioretinal atrophy in macular area.

In Retinal vasculitis due to SLE and in Eales disease visual outcome was affected by proliferative retinopathy. In tuberculosis vision was improved significantly.

In Toxocariasis vision was not improved due to development of tractional retinal detachment.

CONCLUSION

- Posterior uveitis is a serious disease associated with vision threatening complications.
- Posterior uveitis is more common in the age group of 26-35 years.
- All cases of posterior uveitis is of chronic duration.
- All cases of posterior uveitis are severe in nature.
- Retinochoroiditis is the most common anatomical type comprising more than half of the cases.
- Posterior uveitis due to infectious causes are common than non infectious causes.
- Toxoplasma is the most common aetiology detected in posterior uveitis.
- Toxoplasma is the major infectious aetiology found in posterior uveitis.

- In non infectious causes Vogt Koyanagi Harada syndrome, vasculitis due to collagen vascular diseases are the common causes.
- With prompt treatment the disease is controlled in all cases of posterior uveitis.
- Corticosteroids are the most common drugs used for treatment of posterior uveitis.
- Immuno modulators are used as a steroid sparing agents in non infectious posterior uveitis and retinal vasculitis.
- Despite adequate control of the disease the visual outcome is comparatively poor in posterior uveitis.
- The main reason for poor visual prognosis is the structural damage (chorioretinal atrophy in the macula) caused by the disease followed by complications.
- In posterior uveitis visual prognosis depends upon the aetiology, area of involvement and complications.

PART III

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UVEITIS PROFORMA

Name : age/sex: date:

Phone no:

Occupation:

Address:

Complaints:

1) **Symptoms of anterior segment involvement**

   Pain

   Redness

   Photophobia

2) floaters U/L, B/L
3) Defective vision-onset sudden / insidious

   -painless / painful

h/o joint pain

trauma

surgery

history s/o focal sepsis ENT

Dental caries

h/o contact with TB/leprosy/ pet animals

h/o previous episodes

age at onset

no of episodes

response to treatment

h/o DM/ HT

h/o systemic diseases(rheumatoid arthritis& other connective tissue disorders)

family history similar illness

Other significant history

Systemic examination

Pulse : /min BP : mmHg

Cardiovascular system:

Respiratory system:

Gastrointestinal system:

Central nervous system:

Ocular Examination

UCVA RE LE

BCVA RE LE

IOP RE LE

Slit lamp examination

Anterior segment	RE	LE
Lids edema		
Conjunctiva ccc		
Cornea- KPs fine/mutton fat/pigmented		
AC- cells/flare/hypopyon/shallow ac/Peripheral antrsynaechiae		
Iris-CPN/atrophic patches/iris bombe/koeppes/busaccas nodules		
Pupil- RTL/RAPD/irregular/festooned pupil/occulusiopupillae/seclusion pupillae		

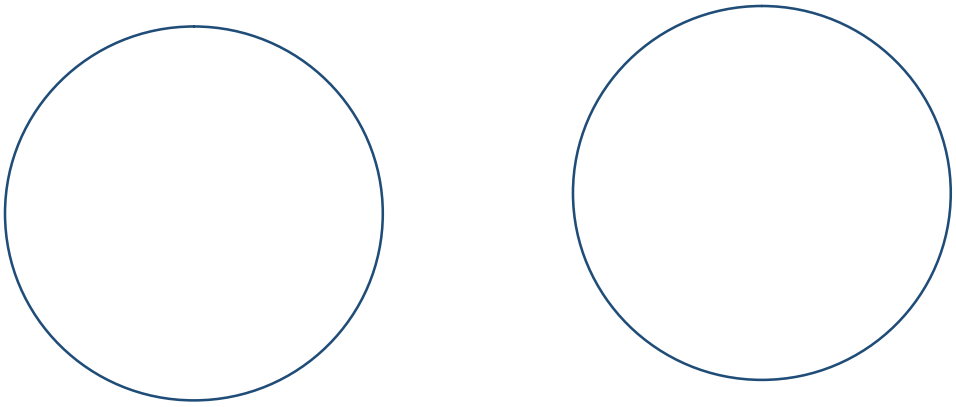
Lens-posterior synaechiae/iris pigment over lens/cataract		
Anterior vitreous-cells/opacities		

Fundus IDO

Fundus	RE	LE
Vitritis grade I/II/III/IV		
PVD		
Choroidits patch-yellowish ill defined patch/atrophic pigmented sharply defined patch		
Retinal edema		
Sheathing of vessels		
Disc edema		
Retinal hemorrhages		

Cystoid macular edema		
Snowbanking/snow ball opacities		

Fundus diagram:RE LE:



Investigations

Fundus photo:

Hb

TC,DC,ESR,Mantouxtest,Chest X ray,RBS,Urinealb/sugar,VDRL/RA factor/ACE/ANA factor and X ray sacro iliac joints

FFA:

B SCAN (if necessary):

TORCH screening

ENT opinion

Dental opinion

Rheumatology opinion

Chest physician opinion

Diagnosis - Acute/ Acute recurrent /Chronic

-Unilateral/Bilateral

-Posterior uveitis

-Aetiology

Treatment

Topical corticosteroids

Topical mydriatics and cycloplegics

Oral corticosteroids

Immunomodulators

Others :

Follow up

BCVA

Intraocular pressure

Healing of leisons

B SCAN (if necessary)

MASTER CHART

S.No	NAME	AGE/ SEX	COMPLAINTS / DURATION	VISION		ANTERIOR SEGMENT		VITREOUS		FUNDUS		INVESTIGATION	DIAGNOSIS	TREATMENT	FINAL VISION	
				RE	LE	RE	LE	RE	LE	RE	LE				RE	LE
1	Pradeep Kumar	25 / M	DV RE -2 Yrs	6/60	6/24	-	Cells1+, Flare 1+	1	4	1f	1d	1	1b	5,2, 1a, 1b	6/60	6/6
2	Rajan	36 / M	DV BE x 1 WK	3/60	3/60	CCC, KP, Cell 2 + , Flare 2 +	CCC, KP, Cell 2 + , Flare 2 +	2	2	2	2	2	2	1a, 1b, 2, 4	6/9	6/24
3	Kuppu	45 / F	F, DV - RE x 1 Month	1/60	6/60	-	-	3	1	6	N	3, 4	6	2, 6	6/36	6/60
4	Girirajan	37 / M	DV BE x 2 Months	6/36	5/60	-	-	1	1	3	3	2	3	2,4	6/24	6/36
5	Varalaxmi	36 / F	F, DV LE x 1 Month	6/6	1/60	-	Cells1+, Flare 1+	1	4	N	1a	1	1a	2,5	6/6	6/36
6	Muniyammal	35 / F	DV BE x 1 Month	HM	4/60	-	-	2	2	4b	4c	1, 6	4	10	3/60	6/60
7	Venkatesan	21 / M	F, DV LE x 1 Month	6/6	6/24	-	Cells1+, Flare 1+	1	4	N	1d	1	1b	1a, 1b, 2, 5	6/6	6/12
8	Sridhar	30 / M	F,DV RE 1 month	6/36	6/6	-	-	4	1	1c	N	1	1b	1a, 1b, 2, 5	6/12	6/6
9	Vijaya	30 / F	DV BE x 2 Wks	6/60	HM	CCC, KP + , Cells ++, Flare 2 +,	CCC, KP +, Cells 2 +, Flare 2 +	2	2	2	2	2	2	1a,1b,2,3,4,9	6/24	6/12
10	Manikam	35 / M	DV LE x 1 Wk	6/18	HM	-	-	1	6	8a	8b	2	8	2, 7	6/18	6/24
11	Rajesh	30 / M	DV LE x 1 Month	6/6	3/60	-	-	1	1	N	7	1	7	2	6/6	1/60
12	Vijayalakshmi	35 / F	F, DV LE x 1 Month	6/6	6/36	-	Cells1+, Flare 1+	7	4	N	1c	1	1b	1a, 1b, 2, 5	6/6	6/12
13	Radha	38 / F	DV LE x 2 Months	6/18	1/2 / 60	-	-	7	2	-	4a	1, 6	4	10	6/18	4/60
14	Uma	25 / F	F, DV RE x 2 Wks	1/60	6/6	Cells1+, Flare 1+	-	4	1	-	1c	1	1b	1a, 1b, 2, 5	6/12	6/6
15	Kathiresan	30 / M	F, DV LE x 1 Month	1/2/60	6/6	-	Cells1+, Flare 1+	1	4	N	1a	1	1a	1a, 1b, 2, 5	6/60	6/6
16	Maheshwari	34 / F	DV BE x 2 Wks	6/60	6/36	CCC, KP, Cell 2 + , Flare 2 +	CCC, KP, Cell 2 + , Flare 2 +	2	2	2	2	2	2	1a, 1b, 2, 4	6/9	6/6
17	Susella	30 / F	DV BE x 1 Month	3/60	2/60	-	-	-	-	5	5	2,7	5	2,7	6/36	6/60
18	Kaniyappan	45 / M	DV LE x 3 Months	6/18	HM	-	-	-	2	N	4a	1, 6	4	10	6/18	3/60
19	Annamalai	40 / M	DV BE x 2 Months	4/ 60	6/60	-	-	-	-	3	3	2	3	2,4	6/24	6/36
20	Palani	29 / M	DV BE x 2 Wks	4/60	4/60	CCC, KP +Cells 2 +, Flare 2 + ,	CCC +, KP +, Cells 2 +, Flare 2 +	2	2	2	2	2	2	1a, 1b, 2 ,4	6/9	6/9
21	Kumari	24 / F	DV BE x 1 Month	3/60	4/60	-	-	-	-	5	5	2, 7	5	2	6/36	6/24
22	Manivannan	31 / M	F, DV RE x 1 Month	2/60	6/6	Cells1+, Flare 1+	-	4	1	1a	N	1	1a	1a, 1b, 2, 5	6/36	6/6
23	Rangan	42 / M	DV BE x 2 Months	6/60	6/36	`	-	-	-	3	3	2	3	2,4	6/36	6/12
24	Mariyammal	35 / F	DV BE x 1 Wk	2/60	1/60	CCC, KP, Cell 2 + , Flare 2 +	CCC +, KP +, Cells 2 +, Flare 2 +	2	2	2	2	2, 5	2	1a, 1b, 2, 4, 9	1/60	6/9
25	Vasantha	28 / F	DV BE x 1 Month	6/60	4/60	-	-	-	-	5	5	2, 7	5	2,7	6/60	6/36
26	Lakshmi	26 / F	F, DV LE x 1 Wk	6/6	1/60	-	Cells1+, Flare 1+	-	4	N	1a	1	1a	1a, 1b, 2, 5	6/6	4/60
27	Rajeswari	21 / F	F, DV RE x 2 Wks	1/60	6/6	Cells1+, Flare 1+	-	4	-	1a	-	1	1a	1a, 1b, 2, 5	6/6	6/36
28	Megala	28 / F	F, DV LE x 2 Wks	6/6	2/60	-	Cells1+, Flare 1+	-	4	N	1a	1	1a	1a, 1b, 2, 5	6/6	6/36
29	Sekar	34 / M	F, DV RE x 2 Wks	1/60	6/6	Cells1+, Flare 1+	-	4	-	1a	N	1	1a	1a, 1b, 2, 5	6/60	6/6
30	Ambiga	31 / F	F, DV LE x 1 Month	6/6	1/60	-	-	-	4	N	1a	1	1a	1a, 1b, 2, 5	6/6	6/60

KEY TO MASTER CHART

RE – Right eye

LE – Left eye

BE – Both eyes

F – Floaters

DV –Defective vision

CCC – Circum Corneal Congestion

Vitreous

1- Normal

2- Grade I vitreous haze

3- Grade II vitreous haze

4- Grade III vitreous haze

5- Grade IV vitreous haze

6- Vitreous haemorrhage

Fundus

1) 1a - Focal yellow white retinal lesion in the macula.

1b - Focal yellow white retinal lesion adjacent to pigmented chorioretinal scar in the acula.

1c - Focal yellow white retinal lesion above supertemporal arcade

1d - Focal yellow white retinal lesion below supertemporal arcade

1e - Focal yellow white retinal lesion superior to macula and below the supertemporal arcade.

1f - pigmented chorioretinal atrophic scar in the macula.

2 - Multiple serous retinal detachments with underlying choroidal thickening and optic disc edema.

3 - Geographic yellow white subretinal patches with indistinct margins from the disc running centrifugally extending to posterior pole and macula.

4 4a- Dense white, well demarcated geographic areas of retinal opacification associated with retinal hemorrhages in the superotemporal arcade and macula.

4b - Dense white, well demarcated geographic areas of retinal opacification associated with retinal hemorrhages in brushfire like extension along the course of vascular arcade with involvement of optic disc and macula.

4c - Granular opacification in the periphery.

5 5a - Cotton wool spots and flame shaped hemorrhages,hard exudates in superotemporal and inferotemporal quadrant.

5b - Cotton wool spots and flame shaped hemorrhages,hard exudates and neovascularization elsewhere in superotemporal and inferotemporal quadrant

6 - Large solitary yellowish choroidal granuloma about 2 disc diameters in size seen in inferonasal quadrant.

7 - Elevated white retinal lesion in superotemporal periphery associated with fibrous band extending to macula.

8 8a - vitreous haemorrhage

8b - peripheral vascular sheathing in the supertemporal quadrant.

Diagnosis

1-Toxoplasmosis a) Macular area

b) Extramacular area

2- Vogt- Koyanagi- Harada Syndrome

3- Serpiginouschoroiditis

4- Cytomegalovirus retinitis

5- Systemic lupus erythematosus with retinal vasculitis

6- Tuberculosis

7- toxocariasis

8-Eales disease

Investigations

1- TORCH screening, Total Count, Differential Count, Erythrocyte Sedimentation Rate
2- Fundus Fluorescein Angiography, RFT, LFT
3- chest x ray and Mantoux test
4- Quantiferon gold
5- Optical Coherence Tomography
6- Antinuclear antibody(ANA), anti double stranded DNA antibody(ds DNA), Rheumatoid factor(RF) and anti neutrophil cytoplasmic antibody (c ANCA, p ANCA)

Treatment

1- Topical corticosteroids, topical mydriatics and cycloplegics
2- Oral prednisolone
3- Intravenous Methyl prednisolone
4- immuomodulators
5- Anti Toxoplasma agents
6- Anti tubercular drugs

7- Panretinal photocoagulation

8- Intravitreal injection Bevacizumab

9- Antiglaucoma medications

10-Anticytomegalovirus drugs

ABBREVIATIONS

CMV	-	cytomegalovirus
VKH	-	Vogt Koyanagi Harada syndrome
IUSG	-	International Uveitis Study Group
ELISA	-	Enzyme Linked Immuno Sorbent Assay
TB	-	Tuberculosis
CNV	-	Choroidal neovascularization
SLE	-	Systemic lupus erythematosus
FFA	-	Fundus Fluorescein Angiography
RPE	-	Retinal Pigment Epithelium
HLA	-	Human Leucocyte Antigen
VDRL	-	Venereal Diseases Research Laboratory
RPR	-	Rapid Plasma Reagin
FTA- ABS	-	Fluorescent Treponemal Antibody Absorption test
HSV	-	Herpes Simplex Virus
VZV	-	Varicella Zoster Virus
HIV	-	Human immunodeficiency Virus

AIDS	-	Acquired Immunodeficiency Syndrome
CT	-	Computed Tomography
IOP	-	Intra Ocular Pressure
ATT	-	Anti Tubercular Therapy
TORCH	-	Toxoplasma
	-	Others
	-	Rubella
	-	Cytomegalovirus
	-	Herpes Simplex Virus
TC	-	Total Count
DC	-	Differential Count
ESR	-	Erythrocyte Sedimentation Rate
RFT	-	Renal Function Tests
LFT	-	Liver Function Tests
IDO	-	Indirect Ophthalmoscopy